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ORIGINAL COMMUNICATIONS.

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THE PRESENT STATUS OF VESTIBULAR TESTS IN INTRACRANIAL CONDITIONS.

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A great deal has been written and said about the merits and demerits of a vestibular examination. Some surgeons and neurologists are enthusiastic about the value of such an examination in suspected cases of intracranial lesion, while others do not find it at all necessary to resort to this method of study. Obviously, no one can state authoritatively just what the present status is. At the University of Pennsylvania, in the service of Dr. Chas. H. Frazier, where practically all intracranial cases are studied neuro-otologically, the same question presented itself. For years the vestibular tests were carried out upon many hundreds of cases with suspected brain lesion. The neuro-otologic examination would be made, the findings and conclusions sent to the surgeon or neurologist in charge and the case lost sight of. Many of these patients drifted away without any opportunity appearing for verifying the diagnosis. Others were operated upon or posted without the knowledge of the otologist so that he was unaware, most of the time, whether he was wrong or right in his premises or conclusions. In order to get some idea of the real place of a vestibular examination, as an aid in diagnosis, Dr. Frazier suggested that the records at the University Hospital be examined for several years back and those cases selected, which were definitely verified either by opera-

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tion or autopsy. This was done by Dr. F. Grant, of his staff, who examined the records and findings in 103 cases. Such a study should throw some interesting light on the real present status of the value of vestibular tests. The cases analyzed were as follows:

1. Cerebellopontileangle tumors, twenty-nine cases.
   (a) Vestibular examination correct in twenty-seven cases.
   (b) Vestibular examination incorrect in two cases.
   Vestibular examination made diagnosis in four cases.

2. Tumors of Fourth Ventricle, three cases.
   Vestibular examination confirmed in three cases.

3. Midline Infratentorial tumors, two cases.
   Vestibular examination confirmed in two cases.

4. Cerebellar Lesions, twenty-eight cases.
   (a) Vestibular examination correct in twenty-one cases.
   (b) Vestibular examination incorrect in five cases.
   (c) Cerebellum ruled out by vestibular examination, in two cases.

5. Cerebral Lesions, twenty-eight cases.
   (a) Hemispheric lesions.
      1. Vestibular examination correct as to side of lesion fifteen cases.
      2. Vestibular examination ruled out posterior fossa lesion in ten cases.
      3. in three of these cases, vestibular examination alone, made diagnosis.
   (b) Pituitary Lesions, thirteen cases.
      1. Vestibular examination correct as to location in ten cases.
      2. Vestibular examination ruled out posterior fossa lesion in two cases.
      3. Vestibular examination incorrect in one case.

It is obviously impossible in the limited scope of this presentation to discuss or cite many of these cases. It might be profitable however, to refer to a few in order to illustrate the types of cases seen as well as the methods of neuro-otologic study.

Case 1: W. O., age 30 years, examined a few months ago, developed tinnitus and difficulty of hearing. Last October, he consulted several otologists in his home town, who treated him with tubal

inflation, etc. The last otologist he consulted found it necessary to do a submucous resection of his septum and remove his tonsils. His tinnitus, deafness and vertigo, instead of improving, became worse. He developed some twitchings of the left side of the face and a little numbness in the left arm. Occasionally, he would have projectile vomiting. As recent as seven weeks prior to our examination he was placed in an institution at home for general study and observation. However, nothing outside of a local ear condition could be discovered. This is recorded to illustrate how faint or few may be the signs of intracranial neoplasm. He continued getting worse and came to Dr. Frazier's Clinic at the University of Pennsylvania. When he presented himself to the neuro-otologic clinic, we found that he had a spontaneous nystagmus in every direction, which at once indicated a brain lesion of some sort. The after-turning responses were all much diminished. The right ear was stone deaf and without responses to the caloric tests from the vertical or horizontal canals. The left ear produced a fair response from the vertical canals, while the horizontal canal produced a good nystagmus and fair past-pointing with a conjugate deviation of the eyes to the left. A diagnosis of cerebellopontileangle tumor, on the right side was easily made in spite of the fact that the vertical canals on the opposite side still retained their function. This was proven several days later when Dr. Frazier successfully removed a tumor from the right cerebellopontileangle. While it is true that at the time of his arrival at the University, there was sufficient clinical evidence to make a diagnosis and that the correct state of affairs was probably known to the surgeon, before he was sent for the neuro-otologic examination, it is nevertheless also true that our examination was purely otologic and our conclusions arrived at independently, were based directly upon the findings obtained from the vestibular tests. What is more, there is no doubt that a neuro-otologic examination, at the very onset of his illness—at the time when the ordinary clinical findings were absent—would easily have made the diagnosis. The patient would have been spared the unnecessary nose and throat surgery and could have been operated on for his true lesion much easier.

Case 2: A. S., illustrates how the neuro-otologic examination was the only means of arriving at the diagnosis. This patient was confined for almost a year in the department for Mental and Nervous Diseases of the Pennsylvania Hospital, without any definite diagnosis. Various neurologists diagnosed it as paresis, or frontal lobe tumor. When we were asked to see her she was totally blind

and stone deaf, in both ears, and unable to walk. The difficulties of an otologic examination in such a patient can easily be imagined. She could not see or hear and was generally intractable. Douching the right ear produced no responses whatsoever. When the left ear was douched, with the head in an upright position, there was no response. When the head was put back there appeared a vertical nystagmus, fairly rapid and active, instead of the normal horizontal nystagmus. We reported to Dr. Strecker, the physician in charge, that the patient had a cerebellopontileangle tumor on the right side. Almost a year later the woman died and an autopsy showed the presence of a large tumor on the right cerebellopontileangle.

Case 3: A. G., age 19 years. Complained of: headaches, failing vision, vomiting; adiadokokinesis, right side; dysmetria, right side; falling to left; marked Romberg; papillitis, three to four diopeters; nystagmus in all directions.

Neurologic diagnosis: cerebellar tumor, side uncertain.

The neuro-otologic clinic reported as follows: "There is a well developed spontaneous nystagmus to the right, left and upward. The vertical canals on both sides do not respond to stimulation while the horizontal canals do—indicating increased intracranial pressure. The nystagmus from the horizontal canal is perverted, suggesting that the lesion is probably close to the brainstem. Turning produces prolonged nystagmus, but almost no vertigo, suggesting brainstem irritation from a lesion posterior to it. Both cerebellopontileangles are uninvolved. The patient has probably a cerebellar lesion involving both hemispheres, but mostly on the left side.

The operative findings disclosed a large gliomatous type of tumor of the vermis extending into each lateral cerebellar lobe and down on left side of pons.

In this case, the diagnosis of a cerebellar lesion was made neurologically, but the otologic examination quite independently, not only arrived at the same diagnosis, but indicated more correctly the extent of the lesion.

Case 4: J. H. P., age 44 years. Chief complaint was pain in the left side of face and head; failing hearing and vision; eighteen years ago injury to left side of face in molar region; eight years ago developed pain in this region and twitching in left corner of mouth; five years ago, hearing became less acute, especially in right ear; two years ago, slow loss of visual acuity, especially when pain is intense; five weeks ago pain in left side of face and head became so marked he was compelled to stop work. Pain is above left eye

and radiates backwards over head and down into left shoulder. Constant tinnitus, left ear; staggers to left, and objects whirl to left; mentality sluggish and all responses delayed; spasmodic tic left side of face; hearing greatly impaired bilaterally; no motor or sensory changes—serology negative; bilateral papillitis 1½ D; fields normal; spinal pressure 10 m.m. Hg.

X-ray: slight clouding right ethmoid sinuses. Pituitary seems slightly enlarged. Teeth negative.

Clinical diagnosis: Optic neuritis toxic origin.

Vestibular examination: cochlear divisions, eighth nerve, both right and left, function normally; past-pointing unduly long but not exaggerated; vertical canals right and left produce no responses to douching; oblique nystagmus on douching left horizontal canal; tendency to conjugate deviation to right from right horizontal canal.

Summary: Prolongation of past-pointing suggests irritation of vestibulo-cerebello-cerebral pathways within cerebellum. Loss of responses from vertical canals suggest increase of pressure against floor of fourth ventricle which is corroborated by oblique instead of horizontal nystagmus after douching left horizontal canal. The tendency to conjugate deviation to right from right horizontal canal suggests a right-sided cerebral lesion. Cerebellum and both cerebellopontileangles may be excluded.

Operation four months later revealed a large gliomatous tumor right frontal lobe.

Case 5: M. F. M. Complained of left-sided headache; vomiting; blurred vision; diplopia on looking down and to right; weakness of left sixth nerve; spinal pressure, 30 m.m. Hg.; papillitis 6 D; fields normal.

Neurologic diagnosis: basilar meningitis unknown origin. Internal hydrocephalus.

Vestibular Examination: Both eighth nerves function in both cochlear and vestibular divisions; vertigo and past-pointing responses normal both ears; quick component of nystagmus to right is interfered with; no reaction from vertical canals both sides.

Summary: Posterior fossa and cerebellum negative. Definite interference with quick component of nystagmus to right suggests left-sided cerebral lesion.

Operation: right subtemporal decompression for relief of pressure.

Patient died two years later. Autopsy revealed large frontotemporal subcortical glioma in left cerebral hemisphere.

In this case the neuro-otological examination definitely indicated the presence of a left-sided lesion as accounting for the increased intracranial pressure.

Case 6: A. P., age 25 years. Complained of headache and failing vision. Four months ago, flashing of colors before eyes mostly on right followed by headache; headaches have since increased; vision is failing and has photophobia. There is diplopia; projectile vomiting; dizziness, vertigo and staggering to right; papillitis 4½ D; lumbar pressure 15 m.m. Hg.

X-ray: some convolutional atrophy of skull.

Clinical Diagnosis: brain tumor suspected unlocalized.

Vestibular Examination: hearing normal, both ears; spontaneous past-pointing right arm, suggesting right cerebellar lesion; absence of nystagmus on douching right ear with head in upright position indicates interference with nerve pathways for eye-movement within right pons; marked past-pointing of each arm to the left and right, suggests that the cerebellum is intact. Exaggerated past-pointing suggests supratentorial lesion irritating the cerebellum by pressure. Pressure from this lesion could account for the absence of nystagmus on stimulating right vertical semicircular canals and for perverted nystagmus from left semicircular canals and for paretic condition of the external recti muscles.

Summary: Ear examination suggests normal labyrinths and eighth nerves, normal cerebellum and brain-stem. Hyperactive responses, chiefly those in past-pointing suggest pressure on cerebellum from above tentorium.

Operative Findings: Puncture of corpus callosum and right subtemporal decompression relieved condition somewhat. Patient discharged. Autopsy a month later showed tumor on posterior aspect of the left occipital lobe of cerebrum. Considerable destruction of brain tissue and cavity had worked forward and had almost broken into left ventricle.

This case showed such a confusion of neurologic data that the only clinical diagnosis possible was a suspected brain tumor with no idea as to its location. The only operation that could be attempted logically, was a subtemporal decompression with a puncture of the corpus callosum. The autopsy later, however, showed that this patient had an occipital lobe tumor located directly above the tentorium and pressing down upon the cerebellum. In other words, exactly where the neuro-otologic examination suggested the lesion to be.

The study of this considerable number of proven cases is naturally helpful in crystallizing vestibular values. As otologists, we are interested particularly in the group of cerebellopontineangle cases. There, the first symptoms are frequently aural and the otolaryngologist is the first doctor consulted. The phenomenon-complex of a totally destroyed ear on one side, with nonresponsive vertical semicircular canals on the side opposite the lesion, and good responses from the horizontal canal on that side, was found to be very reliable. To be sure there were quite a few variations from the typical picture, such as, eight cases, retained hearing on the side of lesion, seven retained function of the vertical semicircular canals of the opposite side; but if one adhered to the principle that a case of this type must show unmistakable neuro-otologic evidence of a central lesion, then the diagnosis can be made with certainty. It might also be mentioned that in angle lesions, the findings obtained in an ear examination are more positive and more reliable than the findings obtained in any other type of examination, but only in this type of case should the otologist maintain his ground even when his opinion does not coincide with the neurologic one. Since cerebellopontileangle lesions are fairly frequent the neuro-otologic examination, even if it does nothing else, serves quite a role in intracranial diagnosis.

Vertigo and past-pointing were found to be reliable indications of the condition of posterior fossa contents. A cerebellar lesion, with very few exceptions, should show impaired vertigo and past-pointing. However, the presence of good vertigo and past-pointing is even more strongly indicative of a normal cerebellum. Another feature of cerebellar lesions is the lack of susceptibility of those patients to vestibular stimulation. Nausea, pallor, sweating and vomiting, as a result of douching and turning, were practically never encountered in cases of cerebellar disease and its presence was used almost as a definite indication that the lesion, if present, is not cerebellar.

No definite phenomenon-complex has been noted for cerebral lesions. Spontaneous, vertical nystagmus may or may not be present; the vertical semicircular canals may or may not respond, the after-turning nystagmus may be shortened or lengthened or be normal, but it has been noted, that cases with cerebral neoplasm can tolerate but very little vestibular stimulation before nausea or vomiting appear. Frequently, neurologic cases require the differentiation as to whether the lesion is supra- or subtentorial. The sus-

ceptibility or nonsusceptibility of the case to ear stimulation can frequently determine that point in a way that no other type of examination can. Thus, given a patient with unmistakable neuro-otologic signs of a brain lesion, who is easily made sick by turning or douching, the case is cerebral. If, on the other hand, large doses of ear stimulation do not upset the patient, the lesion is cerebellar. The presence of a conjugate deviation of the eyes, following stimulation, instead of a full nystagmus is a fairly accurate indication of the side on which the lesion is located.

It must not be imagined that the entire series of cases recorded were like the few just cited. In most of the cases, the neurologic studies disclosed the true nature of the case. It is interesting, however, that the neuro-otologic examination, made entirely independently and without the knowledge of the neurologic findings, and conducted by otologists unskilled in neurology reached the conclusions as shown by the above figures. What is even of greater importance is the fact that in a number of cases, of which some of the reported ones are illustrative, the otologic examination was the only method of study by which a diagnosis could be made. The results of the successes and failures as portrayed by the above analysis state fairly well the *Present Status* of *Vestibular Tests* in *Intracranial Conditions.*

HERPES ZOSTER OTICUS: A CASE WITH INVOLVEMENT OF THE FIFTH, SEVENTH, EIGHTH AND NINTH CRANIAL NERVES AND WITH A COMPLETE VESTIBULAR EXAMINATION.*

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In 1900, Head and Campbell[1] demonstrated the close pathological resemblance between inflammation of the posterior ganglionic chain in herpes zoster and that of the anterior horns of the spinal cord in acute anterior poliomyelitis. This lead to the designation of specific herpetic inflammation as "acute poliomyelitis posterior", which term was adopted by Hunt.

Herpes zoster oticus has been observed by a number of writers since 1871, among whom are Anstie, Green, Blake, Buys, Berger, Vail, Berens and Neumann. But it was not until the exhaustive researches and publications of Hunt[2] that the disease was well understood. His studies, published in 1910, were based on eighty-seven cases of herpes of the cephalic extremity and include a report of nine personally observed cases of herpes zoster oticus.

In Vail's case,[3] presented as a thesis before the American Laryngological, Rhinological and Otological Society in 1906, the brunt of the attack was borne by the upper cervical ganglia; there was no eruption on the auricle and no facial paralysis. The canal had one vesicle on the posterior wall.

There is no doubt that herpes zoster is an infectious disease, although the specific causative agent has not been identified. This question is discussed in an editorial in the *Journal* of the American Medical Association,[4] based on the experimental work of Teague and Goodpasture and of Lipschütz. These observations were published in August and December, 1923. They produced in animals, by inoculations with the virus of herpes simplex, a disease identical with human herpes zoster. They believe that herpes zoster is due to a virus "differing from the virus of herpes simplex only in virulence". They proved that the virus of herpes febrilis can pass along the nerves from the periphery to the brain or spinal cord. Flexner[5] produced encephalitis in a rabbit by inoculation of the

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cerebrospinal fluid from a case of cerebrospinal syphilis; this virus induced vesicular keratitis and dermatitis in the inoculated rabbit and, on cross testing, displays the same immunity reactions as does the herpes virus. These experiments seem to indicate that the infectious agents of encephalitis, herpes simplex and herpes zoster are closely allied.

Emerson[6] saw a case in which the diagnosis of localized encephalitis was made on account of the marked drowsiness. The spinal fluid showed a cell count of 86 with 99 per cent lymphocytes, which suggests the diagnosis of herpes zoster and substantiates Hunt's statement that increase of lymphocytes in the cerebrospinal fluid, because of its frequent occurrence in herpes zoster, would have diagnostic significance in obscure cases. Hunt believes that the so-call rheumatic affections of the eighth nerve may be expressions of herpes zoster of the auditory ganglia without herpes. Likewise, many cases which we regard as rheumatic paralysis of the face, soft palate or even larynx, belong to the group of acute posterior poliomyelitis cases. In Emerson's patient the tonsils and one maxillary antrum were infected and these were the supposed exciting causes of the herpes.

In the published cases, exposure to cold is often given as the etiological factor or the disease is regarded as being of rheumatic nature. In one of Beck's cases he thought repeated attacks of peritonsilar abscess were the source of infection and several writers have removed diseased tonsils because of their supposed causal relationship to herpes zoster. Emerson removed the tonsils and drained the antrum in one of his cases.

PATHOLOGY.

The characteristic pathological changes in herpes zoster are swelling and edema with engorgement of the vessels and small hemorrhages throughout the affected ganglia; degeneration of some of the nerve cells; degeneration of the fibres in the posterior root central to the ganglion.[7] Usually the ganglion of one nerve bears the brunt of the attack, those above and below it being affected to a lesser degree and not sufficiently to cause herpes in their skin zones. Only one side is affected as a rule, although Trautmann[8] reported a case of double herpes auriculae in 1907. It is conceivable that, were the jugular ganglion of both sides affected simultaneously, serious consequencies might ensue.

Typically, herpes zoster oticus is a primary involvement of the geniculate ganglion of the seventh nerve (Fig. 1). From here the inflammation frequently spreads to adjacent ganglia: Scarpa's and

Corti's ganglia of the eighth, the petrous ganglion of the ninth and the jugular ganglion of the tenth cranial nerves. According to Hunt and Cushing,[9] the auricle, auditory canal and tympanic membrane are supplied with sensory fibres from several nerves. The fifth nerve supplies the anterior part of the auricle, the anterior part of the tympanic membrane, canal and meatus. The posterior, mesial and part of the external surfaces of the auricle are supplied by the auricular branches of the superficial cervical plexus; the area between the distribution of the fifth nerve in front and the cervical plexus behind (*viz:* the posterior part of the drum membrane and

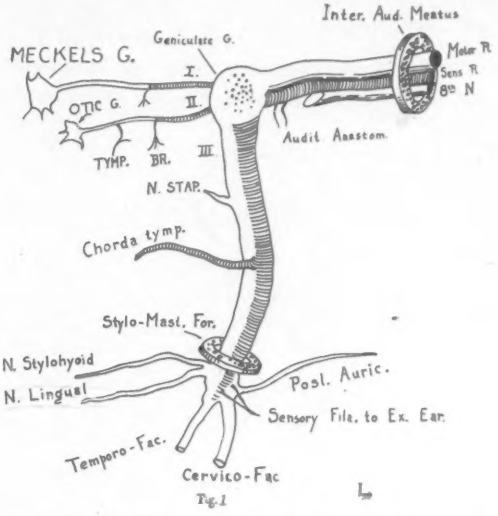

Fig. 1. Diagram, showing relations of the seventh and eighth nerves, geniculate ganglion, connections with Meckel's and the otic ganglia and branches of the seventh nerve. (Redrawn from Hunt's article in Archives of Internal Medicine.)

the external auditory canal, concha, tip of antitragus, antihelix and fossa of the antihelix) is innervated from the geniculate ganglion of the seventh, the petrous ganglion of the ninth and the jugular ganglion of the tenth cranial nerves (Fig. 2). The otic ganglion belongs to the sympathetic system and its nerve cells are multipolar; it is not susceptible to the virus of herpes zoster.

Hunt[10] says: "All the posterior spinal ganglia, as well as the ganglia of the fifth, seventh, ninth and tenth cranial nerves are devel-

oped from outgrowths of the embryologic structure known as the neural ridge. These ganglia are all of the so-called unipolar or spinal type. Peripheral ganglia of the auditory nerve, homologues of the posterior spinal ganglia, take their origin from the neural ridge also, although their cells retain their primitive bipolar character and, in common with other ganglia having this origin, are susceptible to the specific inflammation of herpes zoster. A recent investigation has shown that, in these ganglia, a few unipolar cells are demonstrable in man."

SYMPTOMS.

Most cases have constitutional symptoms for a few days before the eruption appears, such as malaise, headache, fever and aching, together with pain in and around the ear. There may be nausea,

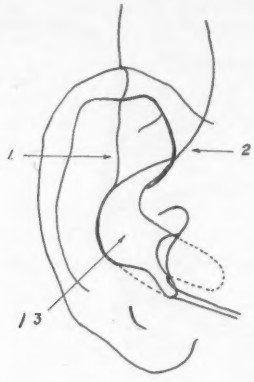

Fig. 2

Fig. 2. Diagram showing area of distribution of the superficial nerve supply of the external surface of the auricle. 1. Area behind this line supplied by the second and third cervicals. 2. Area in front of this line supplied by the fifth cranial nerve. 3. Area lying between lines supplied by the geniculate, petrous and jugular ganglia. (Drawn from description by Hunt and Cushing.)

vomiting and sore throat, depending on the ganglia affected. If the ganglia of the eighth nerve are involved, there will be deafness, vertigo, nystagmus and loss of equilibrium. The eruption is vesicular and is confined to definite areas. The vesicles soon become converted into brownish scabs which eventually loosen and are cast off. Paralysis of the facial nerve is complete in all three divisions and accompanies or follows the appearance of the eruption. Taste on

the affected side of the tongue is abolished. If the ninth and tenth nerves are involved, vesicles appear in the pharynx and larynx and Dan Mckenzie[11] has reported one case with recurrent laryngeal paralysis. In his case the fifth, seventh, vestibular branch of the eighth, ninth and tenth cranial nerves were involved. Herpes was present on the auricle, posterior wall of the meatus, right malar eminence, edge of the tongue, pharynx and larynx. The drum membrane and the hearing were normal. Curiously enough, the cochlear branch of the eighth nerve escaped.

When the cochlear branch is affected, the loss of function may be complete or the evidence of its participation may be only tinnitus and hypoacusis. If Scarpa's ganglion is involved, there is loss of excitability of the vestibular apparatus.

With eruption of the drum membrane, if pain and tenderness over the mastoid are present, together with pain in the ear, and

Fig. 3.

Fig. 3. Diagram to show distribution of eruption in case reported.

fever, otitis media is frequently suspected and the true condition may be overlooked. These symptoms are so suggestive, that it is not surprising that paracentesis is often done.

Recovery is the rule. Sometimes it is fairly rapid, often it is prolonged and occasionally, deafness and facial paralysis persist. Hunt, Jaehne[12] and Muck[13] saw such cases.

TREATMENT.

The treatment is entirely symptomatic. Anodynes for the pain, cleansing and a simple ointment for the eruption are needed. If it can be reasonably suspected that diseased tonsils or sinuses are the source of the infection, appropriate surgical measures are indicated.

Galvanic or Faradic stimulation of the facial area is useful in protracted cases.

CASE REPORT.

I have had the opportunity of studying only one case of herpes zoster oticus: D. J., age 31 years, consulted me on Feb. 1, 1924, complaining of pain in the right ear and dizziness. The family history is negative. He has always been a strong, well man except for measles and mumps in childhood and occasional attacks of tonsillitis. He has three healthy children. Habits are good except he uses a great deal of snuff. Two weeks ago he had something like "grip", with sore throat, aching and fever; after four days there was pain in the right ear and jaw which persisted until yesterday. Simultaneously with the pain he became dizzy and had tinnitus, which are still present. External objects appear to turn to the right. He has vomited twice. Today he noticed that he could not spit straight. Eruption appeared on his ear some time ago but he cannot say exactly when. Recently he has had double vision.

Examination: The patient is a strong, healthy looking man, who must be supported when walking because of vertigo and staggering. There is complete right-sided facial paralysis, involving all three divisions. The right pupil is smaller than the left; the right auricle is swollen and exhibits crusts and vesicles which have the following distribution: tragus, concha and fossa of the antihelix (Fig. 3). The posterior wall of the external auditory canal and the whole of the membrana tympani are covered with eruption. On wiping off the dirty grayish scabs from the drum membrane, the surface is red and moist looking. There is no eruption on the mastoid nor on the posterior or mesial surface of the auricle. The right mastoid is tender; the right side of the pharynx and the right pillars are red and sparsely covered with yellowish white points (ruptured vesicles).

A neurological examination by Dr. F. T. Stevens is, in brief, as follows: "Tremor of jaws, tongue, lips and hands; tongue in midline and protrudes straightly. Pupils react to light and accommodation; no definite strabismus, although the right eye turns out slightly on looking upward or downward; the optic discs are redder than normal, the right being the paler. Deep reflexes increased and equal; no Babinsky, Oppenheim or Chaddock. No sensory loss. No loss of corneal reflex; taste on the right anterior half of the tongue abolished; right facial nerve paralyzed. Joint sense normal."

Hearing: Complete deafness for $C,^{64}$ c and c^4 forks in the right ear; hearing in left ear normal. Weber lateralized to the left.

Vestibular apparatus: Spontaneous nystagmus in all directions of gaze except on looking downward; on looking upward there is a mixed nystagmus which is vertical upwards, combined with diagonal upward to the left. Pointing normal. Romberg: Falls backwards; falling direction changes with changed position of the head. Staggers to the left.

Turning: To the right: good reaction of proper kind of nystagmus, lasting 20 seconds; correct pastpointing; vertigo 8.″

To the left: No nystagmus, no pastpointing; vertigo 2.″
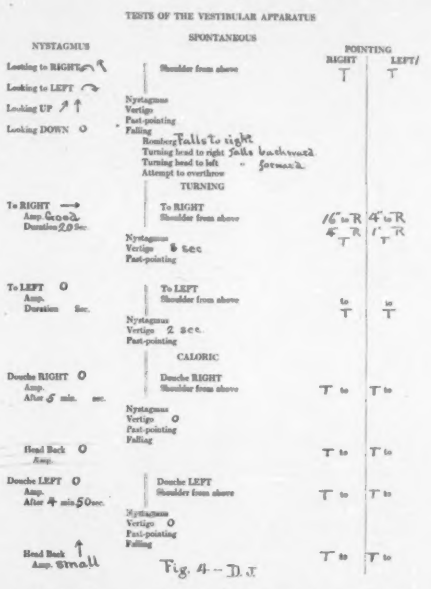
Fig. 4. First vestibular examination. Note the spontaneous nystagmus, absence of reaction in right ear, no reaction from left vertical canals and a perverted reaction from the left horizontal canal. Vertigo response subnormal. It is noteworthy also that, on turning to the left, there is no nystagmus and pastpointing in spite of a good response on turning to the right.

Douching (68°). Right ear: no reaction after 5 minutes.

Left ear: no nystagmus from the vertical canals after 4′:50″; no vertigo; no pastpointing. Head back: a quick, fine vertical nystagmus upward; no vertigo; no pastpointing (Fig. 4).

The patient was sent to the hospital and a paracentesis was done, which revealed a dry tympanum. Cultures from the throat grew chiefly pneumococci. A Roentgen-ray picture of the right mastoid was entirely negative. The urine was normal; white blood count

10,260; no Wassermann nor spinal puncture was made. Temperature normal.

Five days after admission the herpes was disappearing, the right middle ear was dry. At this time he complained that for two days he has had pain and soreness of the right temple and jaw and is tender over the exit of the right supra- and infraorbital nerves. Vertigo and nystagmus have disappeared; Romberg negative.

On Feb. 9, the left ear was douched: no nystagmus nor vertigo from the vertical canals; from the horizontal canal the nystagmus elicited was oblique upward to the right, combined with horizontal to the right of good amplitude; vertigo present. Again on Mar. 24

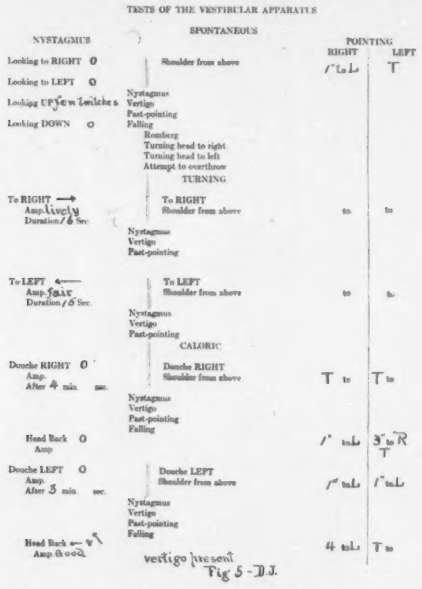

Fig. 5. Vestibular examination two months after onset. The spontaneous nystagmus has disappeared; nystagmus is now present on turning to the left, but on douching there is still no nystagmus from the right ear, none from the left vertical canals and that from the left horizontal canal is perverted. The pastpointing is erratic.

his left ear was douched with the same result. Thirteen days after admission the herpes had disappeared.

On February 26, the hearing in the right ear for all three forks was returning, paracentesis opening almost closed, middle ear dry. A galvanic test of the ears was negative on both sides for a current of 6 m.a.

Ten weeks after admission examination revealed: both drum membranes normal, hearing in the right ear distinctly improved, although the Weber is still to the left. No change in the facial paralysis. A vestibular examination shows some returning function, although there is still no nystagmus from the vertical canals on either side, and from the left horizontal canal the same perversion is present that existed previously; the right horizontal canal is unexcitable (Fig. 5).

In November, eleven months after his attack, a letter from the patient says that he is not doing well; his eyes are all right and the face has "straightened up" but he is still deaf in the right ear and has attacks of vertigo and staggering, with tinnitus.

COMMENT.

This is a typical case of herpes zoster, involving either the geniculate or the auditory ganglia primarily and spreading to the petrous and jugular ganglia, as evidenced by the herpes in the throat and the distribution of the eruption on the ear. It is noteworthy that the anterior wall of the external auditory meatus showed no eruption, which would apparently rule out participation of the ganglion of the trigeminus were it not for the fact that the tragus had a few vesicles on it; the pain and tenderness over the supra- and infra-orbital foramina, appearing some time after the onset, also points to a mild inflammation of the Gasserian ganglion.

The involvement of both branches of the eighth nerve was complete as indicated by total deafness of the right ear, vertigo, loss of equilibrium and nystagmus, and lateralization of the Weber to the left ear. The change in the falling direction according to the position of the head points to a peripheral lesion, but lack of reaction from the right and left vertical canals, no reaction from the right horizontal canal and, especially, a perverted nystagmus from the left horizontal canal is very suspicious of a central lesion. It is, in fact, a typical vestibular picture of right cerebello-pontile angle lesion. This suspicion is strengthened by the information that, eleven months after the onset, he still feels unwell, is deaf, dizzy, staggers and has tinnitus, even though the facial paralysis has recovered.

It is unfortunate that repeated observations of this man cannot be had, but this is impossible as he lives a long distance from Colorado Springs and attempts to get him back have so far been unsuccessful. There is no doubt that he suffered from a typical attack of herpes zoster oticus, whatever other coincident disease may be present.

BIBLIOGRAPHY.

1. HEAD, H., and CAMPBELL: Pathology of Herpes Zoster, Brain 23: 353, 1900.

2. HUNT, J. RAMSEY: Herpetic Inflammation of the Geniculate Ganglion, etc., *J. Nerv. and Ment. Dis.*, 34:73, 1907.
Idem: Paralytic Complications of Herpes Zoster of the Cephalic Extremity, etc., *J. A. M. A.*, 53: 1456, 1909.
Idem: The Symptom-complex of the Acute Posterior Poliomyelitis of the Geniculate, Auditory, Glosso-pharyngeal and Pneumogastric Ganglia, Arch. Int. Med., 5:631 (June), 1910.

3. VAIL, D. T.: Herpes Zoster Auris, THE LARYNGOSCOPE, 16:665 (September), 1906.

4. *J. A. M. A.*, 82:1049 (Mar. 29), 1924.

5. FLEXNER, S.: Experimental Epidemic Encephalitis and Experimental Herpes, Tr. A. Am. Phys., 38:201, 1923.

6. EMERSON, F. P.: Two Cases of Herpes Zoster Oticus, etc., Tr. Am. Otol. Soc., 1923; THE LARYNGOSCOPE, 34:137 (February), 1924.

7. SHARPE, NORMAN: Herpes Zoster of the Cephalic Extremity, *Am. J. M. Sc.*, 149:725 (May), 1915.

8. TRAUTMANN: Ztsch. f. Ohrenh., 53:395, 1907.

9. HUNT, J. RAMSEY: Herpetic Inflammations of the Geniculate Ganglion, etc., *J. Nerv. and Ment. Dis.*, 34:73, 1907. (Quotes Cushing); Arch. Int. Med., 5:631, 1910.

10. HUNT, J. RAMSEY: *Am. J. M. Sc.*, 136:226 (August), 1908.

11. MCKENZIE, DAN: Herpes Zoster Oticus, Combined with Recurrent Laryngeal Paralysis, *J. Laryngol., Rhinol., and Otol.*, 30:339 (September), 1915.

12. JAEHNE, A.: Clinical Aspect of Herpes Zoster Oticus, *J. Laryngol., Rhinol., and Otol.*, 29:333 (June), 1914.

13. MUCK, O.: Neuritis des Trigeminus, des Facialis und des Akusticus, Ztsch. r. Ohrenh., 64:217, 1912.

301 Ferguson Building.

X-RAY OF MASTOIDITIS.

DR. S. G. DABNEY, Louisville, Ky.

Miss Y., age 15 years, very nervous, was taken with an acute suppurative otitis media with bulging of the drum. Free incision was followed by profuse discharge. There were many mastoid symptoms, not only for the first five or six days, but even for two weeks. The extreme nervous character of this young lady made it difficult to ascertain how much actual tenderness was present, and also how severe the pain was. An X-ray examination made about a week after the onset of her trouble showed very slight cloudiness in the cells—repetition of this examination made four or five days later showed no increase in cloudiness. The X-ray was here of valuable assistance in deciding not to operate, and a complete recovery was made without operation.

I could mention several other cases in which the X-ray as I interpreted the report, greatly assisted in deciding against operation. I am inclined to think that unless the clinical symptoms are positive a slight cloudiness of the mastoid cells does not call for operation —it does given an additional reason for further careful observation.

Case 2: A man of about 40, brought in by his physician from a town in the interior of the state. Acute suppuration of the middle ear beginning with tenderness over the mastoid and some fever, together with pain on that side of the head; these were the symptoms that existed two weeks ago. On visiting me practically the only symptoms were profuse suppuration, sagging of the upper posterior wall of the canal, and tenderness on firm pressure over the mastoid; the tenderness was not very marked, and for ten days there had been no headache, no pain and no fever. Because of the duration, however, of the tenderness and continued suppuration with the sagging of the posterosuperior canal wall, I advised early operation. The patient was much averse to this and almost determined to go home immediately. He was persuaded, however, to have an X-ray picture taken, and when this showed extensive destruction of the cell walls he was greatly impressed and agreed to operation, which was done that afternoon. The X-ray in this case corroborated the clinical symptoms and induced the patient to undergo operation which he needed, but it is equally true that operation would have been advised even before we knew anything of the X-ray because of the clinical symptoms—indeed, I would still have

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advised operation (though probably unsuccessfully), if the X-ray picture had been negative.

It seems to me that this brings up a question to which I may refer later—is it always wise to have an X-ray made, even when we have made up our minds from the clinical symptoms to operate? Granting that there are some advantages in this, we must remember the possibility of the X-ray contradicting the clinical symptoms and causing confusion, and we should also remember that to a large number of our patients the additional expense of the X-ray is to be considered.

Besides these two classes represented by these cases I could mention several others in which the X-ray picture had been of material assistance.

Now I wish to refer to a few in which it has been misleading:

Case 3: About three months ago I was asked to see a young lady of about 20, who, after an acute suppuration of the middle ear with rather severe symptoms was still, after two weeks time, having profuse discharge from the ear; great pain localized over the mastoid bone and keeping her awake at night; marked mastoid tenderness, and sagging of the upper posterior wall of the canal. It seemed a typical picture of acute suppurative mastoiditis. I was asked by the family physician and the attending aurist to express my opinion in the presence of the patient. I told her "if she did not have pus in the mastoid bone she ought to have it". I may say that her attending aurist had already expressed this opinion, in which the family physician coincided. My suggestion of having an X-ray picture was agreed to, but without much enthusiasm on the part of my colleagues and, rather odd to say, the family seemed to favor early operation. The financial condition of the family was affluent and the cost of the X-ray was not to be considered. This examination was made about 6 o'clock on Saturday evening. Sunday morning the young lady was taken to an infirmary and an X-ray picture made. To my very great surprise the mastoid was declared to be *free from any disease.* The young lady's menstrual period was going on at the time, and we decided to wait a day or two. The clinical symptoms, perfectly typical of a mastoiditis, continued unchanged. Two days later the mastoid was opened and as soon as the cortex was removed the pus welled up in large quantity.

Case 4: This patient happened to be almost identical with the preceding one. A young lady of about twenty had suffered with profuse suppuration of the middle ear for a little over two weeks. When she reached the infirmary, about 9 p. m., from her home in

the state I found a temperature of about 101°; great mastoid tenderness; pain in the region of the mastoid; profuse discharge, and sagging of the upper posterior wall of the drum membrane. X-ray made the following morning showed only *slight cloudiness of the mastoid cells.* On the strength of this, and thinking perhaps her symptoms the night before had been aggravated by her travel, I postponed operating for two days. Again as soon as I removed the cortex out welled the pus.

I am aware that in both these cases there is a possibility that the pus had developed between the time the picture was taken and the operation, which was forty-eight hours or a little more, but in view of the perfectly typical mastoid symptoms which had been present in both for a week or more previous to my first examination, and in view of the fact that these symptoms continued unchanged for the two or three days preceding operation, I am forced to believe that there was pus in the mastoid at the time of the X-ray picture.

Case 5: A few months ago I saw in consultation with one of my colleagues a boy age 14 years, with an acute suppuration of the ear, which had been complicated a few days before I saw him by paralysis of the external rectus muscle of the same side, and by slight pain in the temporoparietal region; there were no symptoms of meningitis or of brain abscess, nor, as it seemed to me, of mastoid disease. I suggested that it was a case of mild Gradenigo syndrome, and that the boy would recover without operation. A few days later an X-ray picture showed a little cloudiness in the mastoid cells, but as there were no other mastoid symptoms, unless you would so consider those above mentioned (and I do not think they are mastoid symptoms), we decided to wait.

The boy has made a complete recovery without operation.

Case 6: Recently I was asked to see a child with marked symptoms of masotid abscess which had developed suddenly. On reaching the house I found a little girl of about 8 years whose family gave the following history: A week or two previous she had had a little sore throat with some swelling in the glands of the neck. She seemed to make a good recovery from this and had gone to school on the day before I saw her. She had been sent home because of earache. The tisues back of the ear swelled up very rapidly, and an hour or two before I was called the family physician had been summoned. The child had fever of 103,° decided pain about the ear and over that side of the head and was so tender all around the ear and over the mastoid that I found it impossible, even with repeated trials, to tell whether the tenderness was more marked on

pressure about the auditory canal or on pressure over the mastoid. She would scream wherever she was touched in this area. It was impossible to get a satisfactory view of the drum membrane at this time. The auricle was pushed well forward and there was great redness and edema over the mastoid. The picture at first sight presented strong evidences of an acute mastoiditis, and yet I had great doubts of this diagnosis; first, because of the rapid onset of the symptoms; second, because of the absence of suppuration from the ear, though of course we know that mastoiditis can occur without that, and third, the facial expression and general appearance of the child did not indicate to me as grave a malady as mastoiditis. However, I thought it best for the child to go to the infirmary that evening, which she did. At nine o'clock under general anesthesia, on examining the drum membrane and finding it red, I made a free incision into it; there was no discharge of pus for thirty hours, and then it became very profuse; the white blood count was over 33,000; an X-ray picture the next morning showed *evidence of mastoiditis with beginning breaking down cell walls.*

Notwithstanding these laboratory reports I decided to wait a few days. Some simple ear drops and hot ear douches were the only treatment carried out. In three days the fever was all gone, the swelling and tenderness greatly lessened, and there was profuse discharge from the drum. This discharge, however, subsided remarkably quickly, and the child left the infirmary on the sixth day well except for a very slight discharge which has become progressively less. In ten days she was well. It would appear unlikely that there was any mastoid disease in this case. As she recovered the inflammation in the posterior wall of the canal became more evident. Odd to say, in the course of the syringing, the dried remnant of a small fly or gnat was washed out from the auditory canal. The family were certain that this insect had been in the ear since last summer. Can it be possible that this little foreign body in the canal led to misinterpretation of the picture?

Case 7: I mention this case chiefly in the hope that the gentleman through whose courtesy I saw it will give us more complete information than I now have.

Mr. A., age about 37 years, seen through the kindness of Dr. —. When I saw him it was a case of thrombosis of the lateral sinus. A mastoid operation had already been done. The case was one of acute exacerbation of long standing chronic suppuration. The X-ray picture in this case seemed to show sclerosis; some sclerosis was found at the operation, but there was considerable choleasteatomatous mass packed in the mastoid. This raises the question of

whether the X-ray is valuable in the diagnosis between a sclerotic condition of the mastoid and cholesteatoma.

The gentleman made a good recovery from his infected sinus after a skillful operation by the colleague with whom I saw him in consultation.

Of course these few cases—indeed, all of my own experience, would not for a moment permit any final conclusions, but it would appear that much has yet to be learned in regard to interpreting mastoid X-ray pictures. It is frequently said that the picture should be interpreted by the attending surgeon and there is much in favor of this. On the other hand, it should be remembered that the Roentgenologist probably sees such cases from a number of aural surgeons, and therefore would acquire greater experience than any one of them. It would seem to me that his knowledge would be greatly increased if he could be present at each operation in which he had made an X-ray picture; next to this it would appear desirable for him to have a frank and complete report on the operative findings and to keep that along with the picture.

Realizing fully my very small experience I still offer the following as tentative conclusions:

1. In the presence of positive clinical indications a negative X-ray examination should not deter us from operating.

2. This should be explained to the patient or family before the picture is made in such cases.

3. The most valuable report from the Roentgenologist is the statement that the cell walls are broken down.

4. No great importance is to be attached to the report of slight cloudiness of the mastoid cells unless at the same time the clinical symptoms are positive.

5. In doubtful cases X-ray examinations repeated at several days interval may be of much value.

6. In chronic suppurative otitis media it has long been known that sclerosis of the mastoid generally exists. In these chronic cases the X-ray often shows only the sclerosis, and does not inform us as to such other conditions, as cholesteatoma.

7. To understand the relation of the X-ray picture to clinical symptoms, it is desirable to keep the record of operative findings in connection with the picture. Because of his having opportunity to see the work of many surgeons, I think it would be best for the record to be kept by the Roentgenologist, who should familiarize himself reasonably well with the anatomy of the temporal bone; I am aware that this view is not generally held.

911 Starks Building.

A CASE OF CHRONIC PURULENT OTITIS MEDIA WITH SEVERAL INTRACRANIAL COMPLICATIONS.*

Dr. Benjamin H. Shuster, Philadelphia.

Patient, P. G., age 35 years, white, male.

My purpose in reporting this case is not so much to advance something new as to record a series of stormy and unpleasant experiences and to relate some of the puzzling problems that arose and had to be coped with. The patient developed so many complications which centered around a chronic purulent otitis media, that he could well be presented as a small text-book on Intracranial Complications of Otitis Media. The multiplicity of the complications and the method employed to cope with them I thought would be of sufficient interest to merit the presentation of the case.

When the patient first came to my attention he was sent to me by Dr. J. J. Simkins, his family physician, for an examination of his ears, which had been discharging for some time and, what was worse for the patient, his hearing was rapidly failing and had been reduced to the point where it interfered with his normal occupation.

An examination of his ears showed a purulent discharge from both sides with some bad odor; there were polypi protruding into the auditory canals and there were no eardrums left. Hearing was about three feet in the right ear and about six feet in the left ear for the loud voice. He could not hear the whispered voice, Weber to the right and the Rinne was negative, bone conduction being somewhat diminished. It was apparently a conductive deafness due to the middle ear suppuration.

Nose: The septum was deviated but not enough to interfere much with his respiration; there was no free pus. The postnasal space contained some mucus and the nasopharynx was glazed.

There was a chronic pharyngitis, both tonsils were chronically inflamed and liquid pus could be expressed from several crypts.

The thought immediately arose that if the infection surrounding the Eustachian tube could be eliminated the chances for improvement would be better both as to hearing and for the suppuration.

Here the first puzzle arose. The man gave a history of pulmonary tuberculosis that was at present dormant. He also had had tuberculous laryngitis, which had been arrested. The question was, would a tonsillectomy cause a setback in his general condition or not, and was it worth while risking that for a chance of improving

*Read before the Philadelphia Laryngological Society, Dec. 2, 1924.

his hearing. As far as the patient was concerned he was between the devil and the deep blue sea. His hearing was becoming impaired enough to risk losing his employment. After all evidence pro and con was considered, a tonsillectomy was done under local anesthesia, and all precautions were taken not to break down his bodily resistance, by prolonged rest in bed and by giving him sufficient food. He apparently stood this operation fairly well as he lost very little weight. His throat was sore for a week or ten days and then gradually ceased to bother him.

Treatment was next directed to his ears. I removed as many polypi as were visible, employed in turn various local antiseptics, including iodoform dustings, boric acid, mercurochrome, alcohol and various forms of wicks and drains, and vaccines were also tried. At one time I took a smear from the discharge of both ears, after thorough cleansing, and stained it. I found the staphylococci and streptococci and by the Zihl-Nielson method I found the tubercle bacillus. I took precautions to rule out the smegma bacillus by acid decolorization, but the germ was acid fast. If it were some other acid fast bacillus I do not know, nor do I venture to say that the bacillus was responsible for the ear condition, because I am not certain. The germ may have been forced into the ear through the Eustachian tube and lodged there without taking part in the causation of the otitis; or it may have had its share of responsibility. At any rate I used chalmoogra oil for some time without perceptible benefit. Local treatment to his nose and Eustachian tube was not overlooked and carried out faithfully.

The result of the treatment was not satisfactory, his discharge continued, polyps reformed and his hearing was getting worse.

Vestibular tests showed markedly diminished vertigo and nystagmus from all canals, both to rotation and caloric tests. There were no perverted responses nor were there any spontaneous reactions.

X-ray examination of both mastoids showed no breaking down of bone, but sclerosis on both sides.

Treatment was continued for a month or so when the patient received his next complication. About the end of June, 1924, he took sick suddenly with what appeared to be an attack of acute indigestion. He was suffering from nausea, occasional vomiting and felt dizzy. He was under the care of his family physician who, because of his dizziness, asked me if the ears might have anything to do with his illness. I called on the patient at his home. He complained of nausea, had vomited several times and felt dizzy, his dizziness being worse when he moved his head. To me his dizziness and nausea meant more than indigestion; I had but to look at

the patient's position in bed, fearful of moving his head and glancing at me sideways through the corner of his eyes, which showed nystagmus; all of these signs informed me that we were dealing with something grave. His nystagmus was vestibular in character, the quick component to the left side. He slept on his left side and refused to move his head or eyes for fear it would make him dizzy. It is here worth while to emphasize the fact that no patient with a chronic purulent otitis media, when taken acutely ill, can have any diagnosis made of any ailment until ear complications can be ruled out. Here was apparently an innocent case of acute indigestion which turned out to be a labyrinthitis, one of the gravest conditions with which we have to deal. It might have been any other condition which starts with a little fever, headache, nausea, etc.

The patient was in bed a week while his symptoms of nausea, dizziness and nystagmus were gradually subsiding. At about the end of ten days a slight paresis of the facial nerve on the right side was noticed. This lasted for a few days and then slowly began to disappear.

The question now was, what should be the next course to be pursued. Should a radical mastoidectomy be done and probably drainage of the labyrinth be instituted at once or should I wait until the inflammation of the labyrinth should get into the latent stage, when it would be safer to operate? I feared the infection of the meninges while operating during the active stage. I consulted my chief, Dr. Coates, who agreed with me that it would be safer to mark time for a while and then do whatever surgical procedure might be indicated. The fact that it might be a serous labyrinthitis with a possibility of complete recovery without operation was not to be overlooked.

The patient was out of bed in about three weeks after the onset of this last illness. He complained of slight ataxia and that his vision was somewhat impaired. There was occasionally present some slight nystagmus, which I thought might account for what he thought was imperfect vision. He was gradually gaining strength, his facial paresis has completely disappeared and my thought was to at least do a radical mastoidectomy as soon as the patient recuperated. The rotation test elicited a very slight response from the canals on both sides. There was slight nystagmus as well as vertigo.

This plan was soon spoiled by what occurred next. The patient developed an attack of acute mastoiditis on the opposite side, which needed immediate attention. A mastoidectomy had to be done and I was not unmindful of the patient's general condition and particularly had in mind the limitation of the patient's resistance. To do

a radical mastoid operation on both sides at the same time was more than what I thought the patient could withstand.

I consulted Dr. Coates and he advised that a radical operation be done on the acutely inflamed side under local anesthesia. This we did together and the patient got along quite well. His wound healed and the discharge on that side gradually diminished; the other side, that is, the one in which there was a labyrinthitis, apparently did not annoy him and he went to the country for a rest.

He was there for about two weeks (middle of July to early in August) when he began to complain of some headache, mostly over the vertex and frontal region. A few days later he was brought home by the family because his headache became very severe and they thought that occasionally he talked irrationally and could not articulate certain words.

When I examined him I did not observe any aphasia, but he complained of severe headache and his pulse was slow and regular. It was as low as 58-60 and there was no fever. Dr. Samuel Loewenberg, the medical consultant, thought that the patient was developing a meningitis, basing his diagnosis on the slow pulse and the headache, which are often present in the early stages. He thought that it was probably tubercular because the patient was tuberculous. His depressed appearance together with the headache, slow pulse, absence of any chill or chilliness or rise of temperature and the lack of mental alertness or excitability inclined me to the diagnosis of brain abscess rather than one of meningitis. There were, however, no localizing signs of abscess, though there was a suspicion of Kernig's sign and Tache cerebrale. I had the patient admitted to the Medico-Chirurgical Hospital and then arose the most difficult and pertinent question—what next?

The patient was admitted to the hospital, Aug. 11, 1924, and notes made by Dr. Hamlin, the resident Otolaryngologist, state that he was mentally confused, deaf, and was unable to express himself. Dr. Hamlin had the patient under constant observation during his stay at the hospital. He obtained various consultations, and made many important notes, which I freely incorporate in this report. His assistance in the ward and in the operating room on this case were to me invaluable, and I take this opportunity to express my appreciation to him.

Dr. Mershon examined the patient's eye grounds and found them entirely negative.

A neurological examination was made by Dr. Patten, Aug. 12, and the following is his report: "Ocular movements full, pupils react to light and convegence. Right leg a little more flaccid than

left, the foot at rest being in eversion, no ankle clonus or Babinski. Right knee jerk slightly increased over left. There is a suggestive Kernig both sides. Grips hand well; right biceps and triceps slightly increases over left. There is found a slight rigidity on forward and backward motion of the head. Tenderness is found on pecussion of skull over right parietal and left mastoid area.

Findings: There seems to be some difference between the right and left side, but it is not sufficiently marked to indicate a definite lesion. The patient is extremely toxic and mentally confused, therefore, co-operation is reduced to a minimum. The history of mastoid infection on the left side with a discharging ear on the same side at the present time plus the indefinite signs on the right side of the body would point to the left cerebrum as the seat of irritation and probable location of any lesion.

Recommendation: Spinal puncture and cell count and if the cells show a predbmination of polynuclears it might indicate a meningitis more or less localized. If the cells are lymphocytes you would consider the case a toxic meningitis."

The inference from this report would be that, if any search was to be made it would have to be done on the left side—that is, the previously operated side. To my way of thinking, it appeared the other way around. I felt that if any intracranial involvement took place it would occur rather through the nonoperated side than the one which was operated and cleaned; and, furthermore, the greater flaccidity on the right side which was slightly apparent—I thought more proper to interpret as greater spasticity on the left side, which would more likely be an earlier manifestation than flaccidity in a subcortical cerebral lesion. This assumption later proved to be correct. At any rate, the indications, at present, seem to be to drain the source of infection plus a decompression through the mastoid wound and if nothing definite could be located during the operation, prepare the wound so that at the slightest sign of localization it would be but a few minutes work to search the brain. The previously operated side had a large dural exposure which could also be utilized in case localization took place on the other side.

Accordingly, I did a radical mastoidectomy on the right side, under local anesthesia and found sclerotic bone almost to the bottom of the cavity where considerable softening of the bone, channels of pus, and an extradural collection of pus were found. A large part of the tegmen of the antrum and tympanum was removed as well as the sinus uncapped and considerable bone removed anterior to the sinus over the cerebellum. Outside of the localized meningitis there were no local signs, such as bulging, that would indicate brain

abscess. In the presence of so much local infection I did not feel justified in searching the brain and if nothing were found, open avenues of infection into the brain. I left the wound open and decided to mark time.

Meanwhile the following laboratory work had been done:

Urinalysis: negative. Blood count: leucocytes 11,000, small lymph. 24, large lymph. 2, trans. 1, poly. 73. Spinal fluid globulin present, no leucocytes, R. B. C. present. Positive culture staphylococcus albus. Wassermann negative. The laboratory thought that the presence of staphylococci was a contamination, but the same germ was found twice, subsequently.

The following were the notes made by Dr. Hamlin after the operation.

Aug. 13,. 1924. On the day following the operation the patient seemed less confused and more communicative.

Aug. 14. Patient rested comfortably and seemed brighter.

Aug. 15. Another specimen of spinal fluid was obtained, also a blood culture made. The report of this date was staphylococcus albus in the spinal fluid, blood negative. At the time of obtaining the specimen of blood, 25 c.c. of 1 per cent mercurochrome was given intravenously. The patient showed a reaction to the drug as a chill was followed by a rise in temperature and pulse rate with marked diarrhea the following night.

Blood examination: Leucocytes 20,000, small lymphs. 18, large lymphs. 7, trans. 2, poly. 72.

Aug. 17. Patient talking incessantly, hands had to be fastened to keep from pulling on the dressing, asked for water frequently, intake 68 oz.

Aug. 18. I noticed that during his restlessness the pulling on the dressings was chiefly done with right hand. He kicked more with right leg, and there was a decided flaccidity in the previously spastic side, in both arm and leg. I was then convinced that there was subcortical pressure in the temporo-sphenoidal lobe on the right side, that is, the side just operated on. The patient was again taken to the operating room and without any anesthesia the wound was reopened and there was bulging of the temporosphenoidal lobe. Three stabs were made, the last one forward over the tegmen tympani and a gush of bloody, thick pus came out under high pressure. This was drained with a wick of gauze.

From the patient's condition, his progress, the presence of brain abscess and particularly his meningitis, it was apparent that he was hopeless. The report of an occasional success with subarachnoid

irrigation prompted me to institute this measure in hope of even the slightest chance. Surely I could do him no harm.

With the assistance of Dr. Hamlin and Dr. Noble, the resident on our service, I trephined in the right frontal region, entered the cisterna magna through an occiptal-atlantal puncture, while Dr. Noble did a lumbar puncture, and I irrigated with about 100 c.c. sterile Ringer's solution. The solution was kept at body temperature by the nurse in a water bath. In this procedure the technique described by Dr. Eagleton was followed. A specimen of the fluid was sent to the laboratory for culture. Report: Staphylococcus albus a 100 cells per c.m.

Subsequent notes by Dr. Hamlin:

Aug. 19, 1924. Patient appears brighter and recognized visiting physicians, he moves his right arm considerably but his left is quiet, moves both legs frequently. The evening of the 19th the patient was found out of bed standing in the ward. Intake of fluids 44 oz. for the day.

Aug. 20. Patient recognizes attending physicians and his condition seems encouraging. Temperature 101°, pulse 108, respiration 22. Intake of fluids 72 oz. Blood count: erythrocyte 4,280,000, leucocytes 22,150, small lymphs. 15, poly. 85.

Aug. 21. Temperature 101,° pulse 108, respiration 32. Patient's breathing is laborious in type, does not recognize anyone, moves about constantly in bed, muttering in a low voice. In afternoon of same day, temperature 104.6,° pulse 160, respiration 44. Rales increasing throughout chest.

Aug. 22. Patient died, 4:20 a. m. Cause of death, meningitis (staphylococcus albus). All spinal fluid specimens obtained after death were turbid and under pressure. Lowest pulse rate was 58, noted Aug. 15, before murceurochrome was given. Daily remissions of temperature ranging from 98 to 102,° with highest elevation at 6 p. m., until the afternoon before death, when the temperature kept steadily rising, when it reached 104.6° just preceding death.

Pus obtained from brain abcess showed streptococci nonhemolyticus and pyocyaneous.

Spinal fluid injected into guinea pig—report negative for T. B.

Post-mortem: No complete autopsy was made, but Dr. Hamlin enlarged the cranial wounds and found necrosis of the entire temporal bone, which was infiltrated with pus, including the petrous portion. The abscess cavity was as large as a walnut, the brain was softened about the entire region of the temporal bone and the fluid was thick, although not cloudy. The opposite side was negative as far as intracranial complications were concerned.

1717 Pine Street.

OPTIC NEUROPATHY AND SPHENOETHMOID DISEASE.*

Dr. Clifton M. Miller, Richmond, Va.

The literature on sinus disease as a cause of optic nerve disturbances is so voluminous already that necessarily there is great hesitation before one adds further to the subject. However, something may always be learned by a thoughtful consideration of work that has been done and a constant review and discussion of this work may add further knowledge.

The difficulty that we have labored under is that there is almost a complete absence of autopsy reports in this character of cases. It is rather surprising that the connection between nasal pathology and disturbances of the eye functions was not sooner given more attention in this country. In the *American Journal of Ophthalmology* for June, 1887, there is a translation of an article by Ziem, of Danzig, in which he reports visual disturbances as a result of the use of the electro-cautery on the mucosa of the inferior turbinate. The same journal for September, 1895, contains an article by Adolph Alt, in which he refers to this report of Ziem and reports similar cases of his own.

In 1886, Ziem reported cases of restriction of the visual fields as a result of diseases of the nasal accessory sinuses and made the observation that sphenoidal sinus disease produced this condition much more frequently than disease of the antrum. Killian, in 1887, made the same observation. The cases reported by both of them showed gradual improvement of both the visual fields and central vision as a result of draining the affected sinuses. In 1889, Ziem reports a series of seven cases showing similar results.

Had these reports been kept before us an earlier working out of the place of the nasal accessory sinuses as an etiologic factor in eye pathology might have gone forward step by step with our advancement in knowledge of the physiology and anatomy of the nose and the relations of the accessory sinuses to neighboring cavities.

Had this knowledge been worked out many eyes that have lost useful vision would now be functioning and many people would

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have been saved the frequent injections of tuberculin and other agents in the hope that some latent infection would be reached by this method. Much work of a superior kind has been done in the line of determining the place that nasal pathology bears as an etiologic factor in ophthalmic pathology, but much more remains to be done and the more closely the association between the workers in these lines is drawn the more surely and promptly will the results redound to the welfare of mankind. The pathogenesis of glaucoma is still to be worked out and one of its causes will most probably be found in work in this line.

Onodi, in 1908, published the results of his extensive work on the relations of nasal accessory sinuses and the optic nerve, and to this work much of our present advanced knowledge is due. In his very extensive bibliography are found but two or three names of American observers. The valuable contributions to the subject by American investigators having been made since that time. His plates and their accompanying text have much that is valuable to every department of this study. The explanation of the reason for contralateral or bilateral disease of the optic nerve in onesided sinus disease is very impressive and a study of the plates brings the conviction that he is right in his conclusions. In concluding his article he says:

"I wish to make a supplementary statement. I have, in general, called attention to the latent foci and their explorations, but must emphasize that fact that in those cases of sight derangement, in which the oculist cannot confirm the cause, and suspicion of nasal connection exists, even though the finding be negative, it seems well to take explorative measures, and to seek the cause, whether or not the presence of latent foci is suspected. The good success of Schmeigelow and others strengthen us in our belief. I have never failed to use this test in cases given over to our care, and if I was not always successful in locating latent foci, at least I felt nothing had been neglected. I herewith close my clinical observations.

"The casual connection of a onesided, doublesided or contralateral sight impairment and blindness in diseases of the accessory cavities, particularly those of posterior ethmoid cells and sphenoid sinus, is conclusively proven, the anatomic foundation of this theory is sufficiently established by reason of my morphologic findings; they allow of every natural conclusion, without making use of any forced or improbable hypotheses. An exact pathologicoanatomical foundation is still to be created.

"I am convinced that a complete elaboration of this theory would be made possible by exact clinical observations, and through necroscopic investigations, that mutual rhinologic and ophthalmologic researches will close this important and interesting chapter of medicine."

We have proceeded far since this was written but our work has advanced along just the lines pointed out. The work of White is outstanding and his articles are of the greatest importance to anyone who desires to do work in this line. In 1920, he said in *Journal A. M. A.*, "There are various types of accessory sinus blindness, some of which will recover spontaneously, while others will result in permanent loss of vision unless prompt and proper attention is given. Etilogically they are generally divided as: *1*, those due to a direct spreading of the infection to the sheath of the optic nerve; *2*, those due to the toxemia from infection in the sinuses; and *3*, those due to hyperplasia."

Those familiar with the anatomy and pathology of the nasal accessory sinuses can have no doubt of the correctness of his views as to the etiology above given, but there no doubt is sometimes, particularly in the acute cases, a direct choking of the nerve in the optic canal caused by infiltration from the neighboring acutely infected sinus or a phlebitis produced by infection from the sinus which causes swelling of the nerve head. The probability of these two is borne out by the gravity of the prognosis when the size of the optic canal is small, as shown by X-ray plates. It is exceedingly unfortunate that the technique of making the X-ray plates for measuring the size of the optic nerve canal is a very complicated one and few X-ray operators have perfected themselves in it. It is to be hoped that White's technique will very soon be available to all roentgenologists.

In the diagnosis of these cases every means for systemic and local examinations should be used and even then a diagnosis by exclusion has sometimes to be arrived at and what is in reality an exploratory operation performed. A completed serological and physical examination, as well as a most searching history taking is essential. The possibility of a retrobulbar neuritis from a focus of infection far removed, or alcohol, tobacco, quinin or any other drug that may be the cause of amblyopia must be kept constantly in mind. Syphilis here, as in every other organ of the body, is a frequent etiologic factor and may manifest itself either as in active neuroretinitis or in a retrobulbar neuritis with an opthalmoscopic picture that is practically negative.

Woods and Dunn report an analysis of eighty-six cases of optic neoropathies in which syphilis was the cause in 40 per cent of the cases and sinus disease in 12.7 per cent.

They say further, "The acute cases observed secondary to sinus disease all gave the clinical picture of a retrobulbar neuritis without ophthalmoscopic evidence of inflammation or elevation of the papilla". This latter is not the experience of the majority of others who have reported cases, there being frequently observed marked swelling of the optic papilla.

X-ray examination of the sinuses may show involvement, but a negative plate or series of plates taken in six or more different positions of the head does not of necessity rule out the possibility of sinus infection nor decide against operation. The X-ray examina- should include plates showing the sella turcica in order that the size of this may be carefully considered in the neurological examination of the case. Nasal examination may reveal pus in minute amount, a careful tracing of which to its source will lead us to the offending sinus. Every diagnostic means at our command for nasal examination should be utilized. Most frequently shrinkage of the nose with cocain and adrenalin shows no pus or abnormal secretion, but a middle turbinate that will not shrink and is crowded against the septum, which may be deviated high up. This condition is, in the opinion of Skillern, a very suspicious one and should be corrected. That the narrowed condition of the upper region of the nose is upon the opposite side to the optic nerve involvement in unilateral cases is no indication that defective drainage is not responsible for the condition, and this condition must be dealt with as though it were upon the side of the defective eye. The closed condition of the nose may be an indication of sinus disease that has no other symptom that can be discovered by any means of diagnosis now at our disposal. Van der Hoeve says, "The rhinologist cannot say with absolute certainty that a person has no sinus affection". With this statement we, no doubt, all agree.

In the examination of the eye alone factors to aid in the diagnosis may be present or absent. In connection with the ocular examination it must be remembered that intraocular pathology has, in practically every case, its etiologic cause in some location away from the eye. In retrobulbar neuritis and usually in papillitis and optic atrophy the ophthalmologist has no symptom in the eye alone that will allow him to say positively what is the cause of the disturbance in function or abnormal appearance of the nerve head.

In a patient presenting with blurred or decreased vision the ophthalmoscopic examination may or may not show changes in the nerve head where the cause is a sinus disease; such interference with vision, however, is frequently the symptom that causes the patient to seek relief, hence the ophthalmologist is usually the one first consulted. In the absence of any visible signs of optic nerve involvement the symptoms of retrobulbar neuritis are sought.

According to Bordley the most common alteration in the field of vision is an enlargement of the blind spot of Marriott. This enlargement is of spindle form and gradually overspreads a preceding and advancing color scotoma. Narrowing of the field for red is one of the earliest symptoms in the opinion of the majority of observers. Unilateral retrobulbar neuritis points to sinus disease as a cause, but is by no means to be taken as certainly indicating such a cause any more than bilateral involvement indicates the absence of sinus disease. Pain is not characteristic. There may be a typical pain, such as is usually suffered in asthenopia or intense nonlocalized headache, but no characteristic pain is the rule. Careful inquiry into the location, time of onset and character of pain should always be made, but the information elicited by this means will usually not be sufficient to be of real value.

Pain, blurred vision and a semidilated pupil are early symptoms to be given full weight, but the same occur in glaucoma. There is nothing absolutely characteristic of sinus disease as a cause of defective vision to be gotten by examination of the eye alone. The field of vision is the point to be most carefully considered in this part of the examination and variations in the size of the field defects have some significance as well as variations in the visual acuity. It is not unusual for a patient to see more acutely at one examination than another. This is particularly true of the slowly progressing retrobulbar neuritis cases whose vision undergoes marked changes from various extraneous causes, such as changes in atmospheric conditions, or is markedly improved by a free epistaxis. When a patient presents a history in which such statements as the above are contained they must be given due weight and suspicion of the accessory sinuses is strengthened.

The discovery of some other factor such as alcohol, syphilis, etc., as a cause of retrobulbar neuritis does not mean that the sinuses are freed from suspicion, for they may be acting as a contributing cause and the case may not yield to treatment of the apparent cause, in which case the sinuses should receive the same attention as though no other cause had been found.

Conversely, if a diseased sinus is apparent in a case of retrobulbar neuritis or papillitis while the sinus should receive immediate and adequate drainage, still other conditions that may be contributing factors to the optic neuropathy should be sought for and, if found, given appropriate treatment. There should be emphasis put upon this phase, it seems to me, for we are prone to cease further search when a factor sufficient to fully account for the symptoms present is found.

The treatment of these cases does not always involve operation on the sinuses; when the middle turbinate shrinks readily following the application of cocain and adrenalin and no pus is evident after suction, they frequently are cured by a careful regulation of the diet and all the habits of life. There should, at the same time, be local treatment of the nose by shrinkage and suction, followed by the liberal use of argyrol solution, 40 to 120 grs. to the ounce. The 40-gr. solution seems to meet with the approval of many workers in this field. In my own hands the stronger solutions have seemed more effective. The argyrol is not used for any antiseptic effect that it may be supposed to have, but for the detergent effect it undoubtedly has upon engorged mucous membranes. Under such a regimen as that just outlined many of these cases will recover, but they are prone to recur after an attack of grippe or very severe rhinitis and patients should be warned that an operation upon their sinuses may be required at any time for the preservation of vision. Where the middle turbinate will not shrink under the use of cocain and adrenalin and continues tightly in contact with the septum, inadequate aeration and drainage of the ethmoid and sphenoid is to be inferred. In such cases if there is also shown by X-ray films a small optic canal immediate operation is indicated, particularly when the case is one of rapid onset of the blindness with or without ophthalmoscopic evidences of changes in the optic nerve. White's cases show that early operation greatly improves the prognosis. The full and complete examination of the patient and elimination of other causes of optic neuropathy is, of course, essential before deciding upon this course.

The above statement is made in full knowledge of what Cushing has said relative to operating on such cases. His handling of White's paper was even, to his own mind, unfair for he said, "It may be unfair to take random paragraphs from a well intentioned paper," etc. White's attitude toward care in diagnosis and elimination of other causes of the condition under consideration is too well known to allow him to be considered reckless. Rhinologists can scarcely

view with patience such strictures even from a man of such ability as Cushing when he does not hesitate to go into the brain or inflate the ventricles for purposes of diagnosis. Here, as elsewhere, in surgery the work should be done by competent operators, but where all else seems lacking an exploratory operation is frequently indicated, and when so indicated is justified. Such an essential to the welfare and happiness of the individual as vision must be preserved if it is possible to do so. The technique of the operation is too well known from the work of Skillern, Sluder, White and others to need any elaboration at this time. They all emphasize the fact that all that is needed is removal of the middle turbinate, uncapping the posterior ethmoid cells and opening the sphenoid. A complete exenteration of the ethmoid is not necessary. Aeration and drainage is the object sought. At this point the history of three cases may be of interest as they all bear upon different phases of the subject.

Case 1: Mrs. W. G. D., age 48 years; first seen Oct. 23, 1922, complaining of reduced vision and a spot before the left eye. Previous history had no particular significance; had always been strong and healthy; has one grown son. She is now actively engaged in charge of cafeteria for a large firm.

Blood pressure normal, examination of kidneys negative, and Wassermann negative. Fundus of eye showed some optic neuritis in the left side. During the time a thorough physical examination was being made vision gradually became worse until it was reduced to light perception. Examination of the nose showed hyperplasia of left middle turbinate with general ethmoid congestion on this side. It did not shrink under applications of cocain and adrenalin sufficient to give thorough drainage to the nose.

On Nov. 5, left middle turbinate was removed and ethmoid cells uncapped. Following this there was a large amount of mucopurulent secretion, though there was no pus found at the time of operation, ethmoid merely red and soft and the mucous lining of the cells very much swollen. Improvement from this time was gradual, but constant, the optic neuritis in the left eye clearing up slowly until on Feb. 2, with her refraction corrected, vision was 20/20 and has remained so since then.

Case 2: Mrs. J. B. A., age 45 years, seen Dec. 24, 1923, complaining of reduced vision in her right eye and inability to use her eyes for any length of time without severe headaches. She had been repeatedly refracted by competent men without satisfactory result. She had recently had thorough physical examination in a neighboring city with findings all negative.

Examination of her fundus shows no change, nasal examination shows some disease of the right antrum and ethmoid, hyperplasia of the left middle turbinate, which does not shrink under cocain and adrenalin. Suction reveals but little secretion, examination of which shows the presence of the staphylococcus albus.

On Jan. 4, her right antrum was drained, right middle turbinate removed and ethmoid cells uncapped. She made uneventful recovery and on Jan.24 her vision, with correction, was 20/20. With proper glasses it has remained so ever since and she has been able to use her eyes with perfect comfort.

Case 3: Mrs. J. P. L., age 38 years, referred to me Oct. 7, 1924, on account of failing vision, which appeared to be due to her nose. She gave history of vision failing gradually for the past year or more, during which time she had been having nasal treatments, local applications and suction, but this had been of no benefit. The vision at present was light perception in each eye.

She has two healthy children, but about a year ago she was operated on for complete hysterectomy, the cause of this she did not give very clearly. It appeared, from what she said, to be on account of pus tubes. Since this time she has been in fairly good health except for vision. Examination of her nose showed very red and big middle turbinate on the right side, impinging against the septum. This did not shrink on use of cocain and adrenalin, but suction gave no evidence of pus. Ophthalmoscopic examination showed optic nerves somewhat gray in appearance, otherwise nothing noteworthy. Physical examination negative except blood Wassermann showed plus four positive. The administration of salvarsan intravenously brought about a gradual improvement of vision to normal, at the same time the nasal condition improved.

These cases are reported not because there is anything new or startling in the history of any of them, but to illustrate types of the classes of cases that come under our observation. Case 1, showing an ophthalmoscopic picture of papilledema, could be studied with the ophthalmoscope as the vision decreased. While the examination into the neurological, serological and general physical condition was being made the vision gradually decreased until only light perception remained and the edematous appearance of the nerve head with obscuration of its outline became more marked. Following operation the same process in reverse was observed until the nerve head was normal in appearance and vision was normal.

There was no pus found at operation, but the entire posterior ethmoid region was red and congested and the removed material

looked almost like splenic tissue. Through an error culture from it was not obtained.

In Case 2 the difficulty with vision had been going on a long time and numerous attempts to correct the vision by competent ophthalmologists had failed to improve either the vision or the asthenopia. Several weeks stay in a general hospital had failed to help in the diagnosis of her trouble; she had been subjected to all types of examination. Though her vision had been reduced for over a year it returned to normal and the asthenopia was relieved following removal of an impacted middle turbinate and uncapping the posterior ethmoid cells. No pus was found but hyperplasia was present.

This type of case is probably more frequent than is now thought to be the case and a careful search for the cause of reduced and painful vision in eyes that still retain enough visual acuity to be useful would bring to light many cases where nasal abnormality was the cause.

In Case 3 the appearance of the middle turbinate was such that I should have unhesitatingly removed it and opened the posterior ethmoid cells and sphenoid-sinus had not the blood Wassermann been positive. With the positive Wassermann, if the vision had not improved under specific medication and the ethmoid region remained boggy, it would have seemed to me that a portion at least of the pathogenesis of the optic neuropathy was in the sphenoethmoid and they would have been attacked in the hope of saving the vision. The congestion in the top of the nasal chamber as well as the vision cleared up under treatment, consequently no operation was necessary. In conclusion, let me say:

1. That the satisfaction in treating these cases will be greatly enhanced if the rhinologist will himself learn the use of the ophthalmoscope, not for the purpose of usurping the place of the ophthalmologist, but to more intelligently follow the progress of the case in all its details.

2. There is a class of cases of retrobulbar neuritis which seem never to progress to actual complete loss of vision or, at any rate, are very slow in their progress in this direction. These cases are never hopeless from the standpoint of restoration of vision as long as there is no picture shown by the ophthalmoscope of optic nerve atrophy.

3. The finding of one cause sufficient to account for the symptomalogy does not justify us in ceasing to search for all the causes

that may be present and remedy them all in our efforts to restore vision.

4. Where no other cause of an optic neuropathology is found after exhaustive physical, neurological and serological examination has been made, exploratory operation upon the ethmoid and sphenoid is justified in the hands of the competent operator.

BIBLIOGRAPHY.

ZIEM: *American Journal of Ophthalmology*, IV, 6, 1887.

ALT: Idem, XII, 9, 1895.

ONODI: Annals Otology, Rhinology and Laryngology, XVII, 1, 1895.

WHITE: *Journal A. M. A.*, LXXIV, 22, 1920.

CUSHING: Idem, LXXV, 4, 1920.

CRANE: Idem, LXXVI, 1, 1921.

WOODS and DUNN: Idem, LXXX, 16, 1923.

WHITE: THE LARYNGOSCOPE, XXXI, 8, 1921.

CUTLER: Idem, XXXII, 8, 1922.

VAN DER HOEVE: Annals Otology, Rhinology and Laryngology, XXXI, 2, 1922.

WESTLAKE: Idem, XXXII, 3, 1923.

WHITE: Idem, XXXIII, 1, 1924.

BORDLEY: *Virginia Medical Monthly*, XLVIII, 3, 1921-22.

YOUNG: *British Medical Journal*, Dec. 30, 1922.

SKILLERN: Accessory Sinuses of the Nose, Third Edition, Some Unpublished Communications.

SLUDER: Headaches and Eye Disorders of Nasal Origin.

American Encyclopedia of Ophthalmology.

CATARRH OR SINUSITIS.*

DR. NATHAN P. STAUFFER, Philadelphia, Pa.

These twelve cases are reported to illustrate the varying symptoms; the difficulties of diagnosing and complications of and the treatment, and some cures of catarrh or sinusitis. Please remember that the word catarrh is the patient's term, not mine.

We find Webster defines catarrh as derived from the Greek, meaning "a run down". So many times we are consulted by our patients for what they term catarrh. Evidently referring to the running nose, or dropping in the throat. Questioning elicits the information there is a constant blowing of the nose, or a dropping in the throat; "a running down".

Naturally the point to be considered is, are we dealing with an acute rhinitis, or a vasomotor rhinitis; or a chronic sinusitis?

If this is an acute rhinitis, it will have a watery or a mucoid discharge, which will change in a few days to a thicker discharge, and clear up under antiseptic washes and proper cleansing sprays of mild oils.

If, however, it is an acute exacerbation of a chronic catarrh, or sinusitis, the mucoid discharge will be tinged with pus, and finally become entirely purulent in character, and this discharge will persist.

It may be blown from the nose so frequently, the patient will use from two to a dozen kerchiefs daily, or it may cause the constant hawking which is so characteristic of a chronic posterior ethmoiditis.

I think we can safely say that any patient who uses several kerchiefs daily after the first two weeks of a cold, or who has the daily dropping in the throat of a thick discharge, can be said to have catarrh, or a better word is sinusitis. It is easier to diagnose in men than women, because ladies frequently are unaware of the postnasal drop and will swallow it.

Indeed they will deny any discharge until a mirror introduced postnasally will bring forth about one-half dram of thick inspissated pus.

Catarrh. To better illustrate the wide variety of symptoms and complications and sequalae, I will present twelve cases. I will not go into all the details of the tests for hearing and treatment, as that is well known to you.

The easiest to diagnose and a common disease is acute sinusitis.

*Read by invitation before the Philadelphia Laryngological Society, Nov. 4, 1924.

Case 1: J. C., age 22 years, white, male, in the midst of an acute rhinitis, was seized during the night with such acute frontal pain, that morphin would not quiet him, so for several nights he suffered, and then was referred to me. I found a badly swollen set of turbinates pressed upon by a deviated septum, both nares filled with glairy mucous and purulent material.

Transillumination gave shadows over both antrum, and no pupillary reflexes. X-ray showed both maxillary cloudy. Patient's parents refused any operation. He was sent to the Presbyterian Hospital, where a blood count revealed 22000 W.B.C. Under ice caps to both antrums, and twice daily dilation of nares with local applications of 5 per cent cocain and 1/1000 adrenalin; accompanied by internal doses of urotropin gr. V.T.I.D., the pain was immediately reduced, so that no morphin was required, and in a week he had recovered; *i. e.,* there is no discharge from nose; absence of pain, no pus; temperature normal; normal blood count. He never had any previous attacks.

Case 2 (see photo No. 1): Quite the contrary was DeW., age 23 years, white, male, single. He had served four years abroad at the front, and while returning home, had an attack of influenza, during which he developed a cellulitis of the right eye. Dr. Holloway was called in consultation by Dr. H. Mac Veagh Brown, and they promptly diagnosed his condition as sinusitis, and had me operate at midnight, urging haste.

I found a swollen and edematous eyelid and pus in the right anterior nares. He was suffering excruciating pain and X-ray showed his right frontals and right maxillary cloudy. Transillumination confirmed this.

I did a modified Killian and a Caldwell Luc, and the patient made a nice recovery in three weeks. About to leave the hospital he developed a septic pneumonia on the right side; then one on the left. This was complicated by empyema and two rib resections. Followed by a pericarditis with effusion and drainage.

Dr. Jopson introduced drainage tubes under local anesthesia into both lungs and pericardium. He was doing nicely until his kidneys refused to function. Death ensued three months after his initial operation for sinusitis. This was the bravest man I have ever seen, although he had all these resection and drainage operations without any anesthetic, with many painful dressings, constantly having three tubes in him, I never heard him make any outcry, or utter any complaint.

Case 3: Another patient, W. G., male, white, age 47 years, was referred to me for deafness and what he termed was catarrh, which he stated "he had had since a boy". This man had no other symptom than catarrh and deafness.

Examination revealed a purulent postnasal discharge, several kerchiefs daily, and he had marked diminution in hearing, in the right ear, that is, same side as his sinusitis.

Transillumination showed a cloudy right antrum and a dark right pupil. At this point, I want to warn you against the weak lights of some transilluminators. Even with a light blonde patient with some weak electric bulbs, you can hardly get a good red pupillary reflex.

Using a strong light, I have found transillumination even more reliable than the X-ray, for I have had X-ray reports which have come back positive only to have a clear puncture. I have never found a dark pupillary reflex without pus. In this man a Myles trocar was introduced under the inferior turbinate (my favorite spot, as I am farther away from the eye) and very foul typical maxillary pus was soon pouring out.

This patient refused operation for a year, and persisted in demanding to be irrigated. It took two irrigations of 1/5000 bichlorid weekly for a year to effect a cure, which treatment markedly improved his hearing. He has not had any discharge for one year.

Patient No. 4 had quite a different set of symptoms.

Case 4 (see photo No. 2): J. W. H., minister, white, male, age 60 years, came from Maine to the medical service, complaining of shingles and leg ulcer. The usual complete routine examination revealed a normal pulse, normal W. B. C. and normal temperature; but a nasal discharge of some years standing, which he described as catarrh. X-ray and transillumination showed left frontal and left maxillary sinuses cloudy, and a modified Killian and Caldwell Luc operation was done.

In a short time he was able to go home cured of his shingles with all their excruciating pain. This man had never suspected his "catarrh" was the foci and the cause of his shingles and leg condition.

Case 5: Another patient, J. M. W., male, white, age 44 years, motorman, came for relief to the Presbyterian Hospital medical service for catarrh and headache. He had normal pulse and temperature, and a normal leucocyte count, normal heart, normal urine. Nasal examination revealed a leftsided nasal discharge. X-ray and transillumination confirmed the diagnosis of frontal and maxillary sinusitis. He was operated upon, Apr. 1, 1918, when a modified Killian and Caldwell Luc operation were done, and on Apr. 13, 1918,

he was discharged cured. For five years he has been free of his catarrh and his headaches.

Case 6: Mrs. B., white woman, was referred by Dr. Chambers, Allentown, June, 1920. This was a grandmother, who was confined to her bed with septic arthritis, and she had had catarrh and headaches for years (using ten kerchiefs daily). Normal pulse and temperature and W.B.C. Transillumination and X-ray showed cloudy frontals and maxillary.

A modified bilateral Killian was done, and a bilateral Caldwell Luc cured her of her arthritis, and she rejoiced in her renewed lease on good health. Examination revealed a purulent discharge still present in her right nostril. A third operation found a central frontal cell which had thick walls on both sides, and which prevented discovery at the original operations, and which had a small communication into the nose.

This was cleaned out, and her recovery was complete. This woman had complete confidence in her surgeon, and submitted to these three severe operations. Otherwise she would still have her catarrh.

It is interesting to note that she developed four years later a kidney condition, for which I referred her to Dr. B. A. Thomas, and she died last year of a septic kidney condition. A further illustration of the harm of permitting these septic sinus lesions to go unoperated upon. For I believe her death was due to a metatasis from her nasal lesion.

Case 7: J. T. Y., age 34 years, male, white, teacher. Hoarseness and attacks of indigestion. Normal pulse, normal temperature, normal W.B.C. and urine and heart. Seldom uses a kerchief, but frequent colds. and a constant clearing of throat postnasally.

A discharge is daily found on the posterior pharyngeal wall, coming from the posterior ethmoiditis and sphenoid region. Refuses operation. Daily irrigation of salt and soda with weekly applications of argyrol 25 per cent, and suction apparatus have relieved him of his indigestion and hoarseness.

Case 8: Prof. B., referred by Dr. F. L. Greenwalt, white, male, age 40 years, married, teacher. Tired all the time, hoarseness, no ambition and use of several kerchiefs daily.

Normal pulse; temperature; W.B.C. and heart, and urine. Examination shows purulent nasal discharge, and transillumination and X-ray show cloudy frontal and maxillary.

Modified Killian cured him of his exhaustion and hoarseness. It is interesting to note in this case that the frontal operation cured the

antrum trouble, still further confirming the opinion that the maxillary antrum was only acting as a reservoir for the pus from the frontal.

Case 9: H. H., male, white, retired banker, age 45 years. Referred from Maryland by Dr. P. L. Travers, because of a nervous breakdown. He had had constantly hawking and blowing of his nose for many years, which had been labeled "the usual catarrh". He has normal pulse, temperature and urine, but could not do any active service, *and was afraid to go anywhere by himself.*

Examination revealed a profuse discharge of pus in his nares, while X-ray and transillumination revealed cloudy antrums. He had been operated upon in New York by Dr. Shamberg for several diseased roots in his superior maxilla.

Under general anesthetic, I did a Caldwell Luc, and this cured him of his nose blowing, and so improved his general condition, that he got rid of his nervousness and also his wife, and he has gone to Europe.

I present tonight *Case 10:* E. J., colored woman, age 60 years, married. Brought to the Presbyterian Hospital complaining of headache, and "feeling funny in the head".

She was supposed to have been cured of a right ethmoiditis years before, as a turbinectomy had been done. She complained of "catarrh".

I found pus in her nares and advised a frontal, as the X-ray showed a cloudy maxillary sinus. She suddenly developed acute mania, and had to be restrained in a straight jacket. Several consultations with Dr. W. E. Hughes were held as to the advisability of operating while in her present condition, as it was feared operation might aggravate it.

After keeping her under observation for a week, and believing her mental condition due to a toxemia, at his earnest solicitation, I did a modified Killian, whereupon her mental condition immediately cleared up. Whether she remains so, remains to be seen. At least she had a very active sinusitis and her mind is clear today, almost a year later.

Case 11: A. V. H., age 65 years, white, male, teacher. Complains of catarrh, inability to breathe through nose, and talks thick and asthma; with postnasal dropping. This patient has many nasal symptoms, yet sinusitis was never diagnosed.

He has a marked purulent nasal discharge with polypii, all probably causing his asthma. He has had two strokes. How much has the chronic sinusitis had to do with causing the weakening and breaking down of his arterial system?

I present tonight *Case 12* — Name, F; age 40 years, is quite the reverse. Here is a patient who never knew he had any nasal condition worth telling, and it was only at close questioning I elicited nasal trouble. His general health has always been excellent, and but one kerchief daily. He had the typical ozeona breath. Normal pulse, normal heart rate, normal respiration, no eye symptoms. Even a frontal sinus operation failed to send his temperature above 100° F., and that only for part of a day, and yet his frontal cells were filled with foul pus.

He went to his physician for an abscess on his forehead which was lanced. It did not heal and a surgeon lanced it again, but Dr. Speese suspected something deeper, and so had me examine his nose. It looked like the typical ozeona, but I found a small amount of pus in each superior nares under the middle turbinate, and thought he had a frontal sinusitis.

This frontal was thought to be only suspicious by the X-ray man but his maxillary was reported cloudy. It was found at operation that nature had been kind to him, and instead of necrosing his internal plate of the frontal bone, it had been burrowed through the thicker right external plate and then crossed and pointed as an abscess over his left frontal about two inches above the right eyebrow. I did a modified Killian on him, and will complete his maxillary later, as he was too cyanotic on the table to finish the operation.

Conclusions: 1 Here are twelve cases with widely varying symptoms. Case 1 had headaches and catarrh. Case 2 had catarrh and eye pain. Case 3 had catarrh and deafness. Case 4 had catarrh and shingles. Case 5 had catarrh and headaches. Case 6 had catarrh and acute arthritis. Case 7 had catarrh and indigestion and hoarseness. Case 8 had catarrh and exhaustion and lack of ambition. Case 9 had catarrh and nervous prostration with various phobias. Case 10 had catarrh and a funny sensation in head which developed into acute mania. Case 11 had catarrh and a closed nose with asthma. Case 12 had catarrh and right frontal abscess pointing on his left frontal bone. All had sinusitis in varying grades, and were either relieved; or cured by proper medical, or operative treatment.

For years their catarrh had been ignored or treated lightly, yet when the sinuses were opened and thoroughly cleansed, the catarrhal symptoms promptly cleared up.

2. Strange how tolerant the heart and temperature were in all these cases to so much pus in the system. Most of the cases had a normal pulse rate, a normal temperature, a normal W. B. C. and a normal respiration, and yet in all of them virulent pus was found and in large quantities.

3. It was peculiar how some had few symptoms, and yet had grave complications, while others had many symptoms, and few complications. It is hard to explain this just on the ground of an acquired immunity.

4. These cases certainly prove catarrh, or sinusitis can be permanently cured.

5. Also that the modified Killian operation cures and leaves the patient as natural as before operation. This does not cause the hideous disfigurement of the radical Killian.

6. Finally it is of extreme importance to question closely the patient, as to all his symptoms and examine thoroughly any nasal complaints in order to cure him.

2015 Walnut Street.

CASE REPORT: PECULIAR CONDITION OF THE UPPER RESPIRATORY TRACT FOLLOWING X-RAY TREATMENT OF CERVICAL ADENITIS.*

Dr. M. C. Myerson, New York.

The following is the report of a case of cervical adenitis which was treated by X-ray and displayed a very stormy and interesting course following the treatment. Whether the child would have been as sick as he was is conjectural. Certainly his local respiratory tract picture must be accounted for by the fact of the X-ray treatment.

R. M., male, age 2½ years, was admitted to the hospital June 19, 1922, and died four days later. He was admitted with a diagnosis of acute pharyngitis and bilateral otitis media. His temperature at this time was 102,° his pulse 100, and respirations 28. The child had had a cervical adenitis of unusual dimensions and had been subjected to X-ray treatment four times within the past two weeks. The last treatment had been given two days before. When seen at home he had an edematous sloughing pharynx. Diphtheria was ex-

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*Department of Laryngology, Kings County Hospital, service of Dr. H. Arrowsmith.

cluded by the consultant and the child was sent to the hospital. Upon admission a thin slough extending from the fossa of Rosenmuller to the posterior pharyngeal wall, uvula, laryngeal structures, and upper trachea was noted. Auscultation revealed a to and fro sound over the trachea, which was evidence of a loose membrane in this structure. There was no evidence of bronchial obstruction. It was advised because of his poor general condition, that the patient be watched closely, with the idea in view of bronchoscopic removal of the membrane, should it become obstructive. The granular, soft, reddened appearance of the mucous membrane was quite unlike diphtheria.

The following day there was evidence of a distinct impairment of transmission of sound through the right main bronchus and evidence of slight transmission of sound through the left bronchus. Some of the slough had evidently accumulated at the tracheal bifurcation, and it was decided to perform bronchoscopy for the removal of the obstruction.

At bronchoscopy the epiglottis was found greatly swollen; it was granulated, edematous, and angry red in color. The arytenoids were similarly involved. The upper half of the trachea was injected; the mucous membrane was thickened and of darker red color. The right main bronchus contained a thick semisolid, viscid, white substance, which was probably disintegrated necrotic membrane. This was removed by suction. The left main bronchus at its tracheal orifice had a similar thick viscid membranous substance flapping to and fro, and partly obstructing its lumen. This was also removed by bronchoscopic suction. There was a distinct improvement in the local respiratory picture after bronchoscopy. The pulmonary sounds had disappeared and the child was breathing much better. On the third day the child became semicomatose, the head was held, rigidly, while the knee jerks were slightly exaggerated. There was a slight Babinsky and Brudjinsky reaction on either side, and a slight Kernig on the left. At this time he began showing signs of a bronchopneumonia. There was beginning evidence of a possible terminal meningitis. The child gradually went into coma and died on the fourth day after admission. The laboratory reports were negative for diphtheria. Direct smears and cultures showed staphylococci, streptococci, and a gram negative bacillus. The spinal fluid on the third day was slightly turbid and under increased pressure. There was an increase of globulin content; 165 polymorphonuclears, and 30 lymphocytes per c.m.m. Wassermann reaction was negative. The colloidal gold curve was indicative of tuberculous meningitis.

An autopsy was obtained. A brief summary of the findings follows:

The mucous membrane of the larynx and trachea was replaced by a light yellowish gray membrane; when washed off, the mucosal surfaces of these structures was somewhat granular. The tonsils, which had been of very large size before the X-ray treatments, were now found to be about the size of a small pea. They could barely be found. The tonsils, pleurae, interstitial lung tissue, liver, kidneys, mesentery, pericranium, meninges and all intracranial structures contained many tubercles. The cervical glands were dissected out; those on the left side were found to be of about normal size; on the right side the glands were slightly larger than normal. On section, these glands showed caseous areas.

From the autopsy it would appear that the mucous membrane of the upper respiratory tract and the tonsils reacted strongly to the X-ray treatment, in addition to the cervical glands, in this case. This was a case of membranous pharyngitis, laryngitis and tracheitis, which followed shortly after the X-ray treatment. Communication with the Roentgenologist who treated the child revealed that the boy was quite ill at the time of his treatment. The Roentgenologist assured that he had used the usual dosage.

This case is not reported as a criticism of X-ray treatment. It is reported, however, to demonstrate that there are individual cases which will not react favorably to X-ray treatment, and therefore great care should be exercised in the selection of these cases for such special treatment. The question arises as to whether the treatment in this case disseminated the tubercle bacilli and caused in addition a peculiar reaction on the part of the mucous membrane of the upper respiratory tract.

30 E. 40th Street.

A PLEA FOR THE MORE GENERAL USE OF LOCAL ANESTHESIA IN TONSILLECTOMY.*

DR. W. HARRY BARNES, Philadelphia.

I was surprised last year to learn that ether is never used by the leading oto-laryngologists of Paris and Bordeaux. I could get no satisfying reason for this. Chloroform is the general anesthetic of the French surgeon. Having had many dangerous experiences with chloroform, in all the operations on the ear, nose and throat, I welcomed the opportunity to learn how they administered it so safely. I will not describe their method here, but the striking fact is that they seldom use a general anesthetic at all. Radical mastoidectomy, radical accessory sinus operations and even total laryngectomy are regularly performed under local anesthesia. I think that their dangerous experiences with their only general anesthetic—chloroform—has compelled them to make their technique of local anesthesia very perfect.

Tonsillectomy in Paris, with a few exceptions, is done under local anesthesia in adults and with *no* anesthesia in children. The children are held in the sitting position by strong orderlies, while the surgeon with a punch forceps (Rualt's) punches out more or less of the tonsils, and scrapes off more or less of the adenoids with a curet. In many adults needing tonsillectomy, they try for months to destroy the tonsils with diathermy, before doing the local tonsillectomy.

Advantages: The *local* operation presents many advantages over the operation under general anesthesia.

1. It makes an operation safe which would be otherwise unsafe: At once we think of patients with diseases of the heart and lungs, in whom a general anesthetic is contraindicated. In this group fall all the tuberculous, who without local anesthesia would be better off with their tonsils, and who bear the local operation well. The same is true of the chronic cardiac cases, muscular and valvular.

2. It makes an operation permissable which would otherwise not be so, such as in case of: hyperthyroidism, nephritis (especially chronic interstitial), uremia, diabetes, hepatic cirrhosis, general debility and lead poisoning. The question of status lymphaticus is as yet unsettled, but the reports of thymic death with general anes-

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thesia far outnumber those with local anesthesia. The same is true of acidosis. It must be remembered, however, that in most of the above conditions, there must be a strong indication for the operation to perform it even under local anesthesia. Such indications would definitely show the tonsils responsible for conditions dangerous to life, or responsible for the condition under consideration (*e. g.*, enlargement of the thyroid from tonsillitis). The judgment of the danger of the following conditions must rest with the surgeon: *a.* recurrent attacks of acute tonsillitis, *b.* chronic tonsillitis causing otitis media, accessory sinus infection, submaxillary adenitis, etc., *c.* chronic tonsillitis with no other cause for endocarditis, arthritis, nephritis, anemia, asthma, etc.

3. It presents a distinct advantage over general anesthesia in the safer performance of the operation, as instanced by the following untoward events: *a.* lung abscesses and postoperative pneumonia were thought to be embolic in origin, but W. F. Moore, of Philadelphia, collected statistics of 200 cases of post-tonsillectomic pulmonary abscess that indicate the aspiration of clots or infective material (quoted from C. Jackson). In local anesthesia, the cough reflex (the watch dog of the lungs) is present and any clots or infective material is immediately coughed up, thus preventing lung abscess and pneumonia from this source. *b.* Hemorrhage. During the operation, blood may escape, and the etherized patient swallow it unnoticed—the first indication of its presence being the vomiting of a large quantity of blood. In local anesthesia the patient always feels the blood, and attempts to expectorate or swallow direct the surgeon's attention to an otherwise unnoticed hemorrhage. *c.* Shock is very much lessened by local anesthesia, so much so that Crile, of Cleveland, uses it often in connection with general anesthesia in general surgery. *d.* The operation is much easier and quicker with the co-operation of the patient, who can raise or lower the head or move it towards one or the other side, open the mouth more or less, expectorate, swallow, etc., all of which helps at times. *e.* All the dangerous conditions, like thymic death, cardiac or respiratory inhibition, etc., have their premonitory phenomena, which in the conscious patient will bring forth some exclamation or movement, which the watchful surgeon will note—thus the respiration and the heart action can be helped before they become weak or cease. *f.* All the unpleasant postoperative effects of the ether are escaped, such as nausea, vomiting, headaches, etc.

4. I have found a large group of patients who will readily undergo the operation under local anesthesia, but who absolutely refuse it

under general anesthesia. It is true that some of these could be persuaded into the general anesthesia, in which case they frequently take it badly and are often shocked. But the most will postpone the operation or refuse it absolutely. The latter class is so ignorant that we are tempted to ignore them and leave them to their fate. But as physicians, we are their custodians and should hold ourselves accountable if any unfortunate termination ensues. The fate of those who postpone it is variable, but conservatively estimated, 25 per cent of them have unfortunate happenings and all are much worse off for the postponement. The local operation in this fourth group should give nearly 100 per cent of perfect cures.

Disadvantages: The disadvantages, for the most part, are imaginary, either from the surgeon's viewpoint or that of the patient. Ignorance of the technique by the surgeon makes him multiply the dangers and hunt up the unusual cases. "Fear is the expression of ignorance" says the old adage. Another surgeon will actually decry a method or an instrument because he is not skillful enough to utilize it, or has had some success with another. When the stethoscope was introduced, the leading internists claimed that the unaided ear was better. If a surgeon cannot perform a tonsillectomy as well under local anesthesia as under general, he does not know the operation well enough to perform it at all.

There is less objection to the local operation by the laity than by the profession, and the many successes of local anesthesia are making the general public demand it rather than fear it. The few patients who object to it claim nervousness or heart trouble. If such really exists, it can be counteracted; and if it cannot is then a *positive* contraindication to a general anesthetic for any operation whatever.

What, then, constitutes the true disadvantages to the operation under local anesthesia?

1. Hypersusceptibility to the anesthetic. This has been the subject of research and study by every scientific body, both here and abroad. The conclusions are so definite and so well known that I will pass them over with only this word. Cocain should never be injected into the tissues. Adrenalin should not be used stronger than 5 drops to 10 c.c. of the solution used: novocain or identical products (procain, etc.) is the safest anesthetic. Its percentage is somewhat important and is dependent upon the condition of the tissues, for instance: 4 per cent may be used if you are sure there is no possibility of entering a vein. It must be understood that I refer only to fresh, potent and properly made solutions of all drugs.

The patient's susceptibility can be measured before the operation by a very simple method. Apply 10 per cent solution of cocain to the nasal mucosa (or any other strength that the surgeon is familiar with). Having examined this mucosa before the application, the surgeon will know absolutely *a.* how the the tissues should react and *b.* that there should be no general reaction, such as acceleration of the pulse and respirations, headache, confusion, twitchings, dilatation of the pupils, fainting, etc. Any general reaction contraindicates the use even of novocain. A local reaction more than twice normal should require further and more careful study before this anesthetic is selected.

2. Hyposusceptibility to the anesthetic. The tests above mentioned will also determine this. Study of the research upon local anesthetics together with the examination of the operative field will determine whether a safe percentage will be sufficiently effective during the time necessary for the operation. In these cases, help is obtained by the internal administration of the bromids, luminal, morphin and scopolamin, if their use is permissibble. Further, the patient may (or may not) be previously told that some slight pain may be expected when the pedicle is severed. (Many interesting studies of the use of very weak anesthetic and nonanesthetic solutions, like sterile water, may be read in the literature.)

3. Obliteration of the landmarks by the anesthetic. This is negligible when a small amount of concentrated anesthetic is used, such as 4 per cent novocain. But as this is not as safe nor as sure, a larger amount of a weak solution (one-half of 1 per cent novocain —1 to 2 ounces) is preferred in my service. The ballooning of the pillars and the tonsilar fossae make it often impossible to use the La Force method with buried tonsils. Even with the dissection method, considerable experience is necessary to be able to distinguish the tonsils from the pillars—the color being the main guide.

Indications: In addition to the usual indications for tonsillectomy, the local operation is indicated in all persons above 14 years of age who will submit to it, but especially in the presence of other diseases which contraindicate a general anesthetic. Among these diseases are enumerated: Tuberculosis and other disease of the lungs (especially emphysema), cardiac disease, both valvular and muscular, nephritis (especially chronic interstitial), diabetes, acidosis, obesity, hepatic cirrhosis, etc.

Contraindications: These include the usual contraindications for: any operation, tonsillectomy, and the true contraindications for the local operation.

1. Some contraindications for any operation are: hemophilia, arteriosclerosis, acute general infections, blood dyscrasias, syphilis, enlarged thymus, mental and nervous diseases (the first mentioned being a *positive* contraindication).

2. Contraindications to tonsillectomy may be grouped as follows: *a.* Acute local infections as lacunar tonsillitis, peritonsilar abscess, stomatitis, postpharyngeal abscess, Vincent's or Ludwig's angina, cervical cellultis, abscess of the cervical lymph nodes, etc. *b.* Anomalous blood vessels: anomalous position of the internal carotid artery, anomalously placed or excessively large ascending pharyngeal artery, very large artery in the anterior pillar, new or misplaced vessels due to previous operations or trauma, etc. *c.* Technical difficulties, such as too small mouths, in children under six months, deformed mouths after burns, trauma, etc., fixation of the tongue by cellulitis, etc., malignant neoplasms anywhere about the mouth, pharynx, larynx, etc. (also a *positive* contraindication), adherent tonsils in seasoned singers who desire a guarantee of the same quality of voice.

3. The true contraindications to the local operation (where the general anesthetic is to be preferred) are: *a.* Children and nervous individuals unwilling or unable to co-operate, in whom the sight of blood, sound of instruments or the feeling of a slight pain would render them unwilling for the completion of the operation. *b.* The aged, or those in whom much hemorrhage is expected. *c.* Where there exists a venous engorgement of the vessels of the head and neck, with the likelihood of getting large quantities of the anesthetic into the blood stream. *d.* Where the patient shows a hypersusceptibility to the cocain group (*positive*).

CONCLUSIONS.

I can say nothing new or original concerning the technique of local or regional anesthesia. I wish, however, to advise those who have not yet successfully used local anesthesia not to study the method noted in some work on throat diseases by a laryngologist. The only safe and satisfactory results can be obtained from studying the works of regional anesthetists, such as Labat in this country, and Canuyt and Rosier abroad.

The disadvantages of local anesthesia in tonsillectomy are practically *nil,* and the advantages of it over general anesthesia are so great that I am sure that when a surgeon once learns to properly apply the local method, the general anesthetic will be used as seldom for tonsillectomy as it is for paracentesis of the tympanic membrane

1315 North 15th Street.

INFECTED TONSILS AS THE CAUSE OF THYROTOXICOSIS.

Dr. H. T. Bailey, Phoenix, Ariz.

When the entire system is surcharged with the products of the thyroid gland, we call the condition thyrotoxicosis. The symptom-complex of Basedow's disease illustrates the condition. While Basedow's disease is a thyrotoxicosis, all thyrotoxicoses are not Basedow's disease.

The tonsils occupy an open position in the throat, that is no food can pass into the stomach, or air into or from the lungs, without passing them. Particles of food and bacteria mixed with mucous will gather on or about the tonsil. This mixture of food, mucous, bacteria, etc., will putrify or degenerate, which forms toxins. This degenerated mass, in turn, lowers the vitality of the tissue where it is lodged, usually the tonsil crypts. With a tonsil crypt spoon, I have excavated and obtained streptococci, staphylococci, pneumococci, catarrhallis, bacillus capsulatis mucosis, Vincent's fusiformis and many others, even almost a pure culture of diphtheria when the patient had never had diphtheria and could not remember of being near a case of diphtheria.

These tonsil crypts may be considered a sump or a catch basin to catch the different offending organisms. These infected crypts inclosing their poison material may be sealed over during any inflammatory process and thus develop a "blind abscess".

It matters not whether the mouths of these crypts are open and exude pus or that they are sealed and have an imbedded abscess, the tonsils are diseased and the toxins are carried into the circulation. It is conceded that these bacteria and toxins cause rheumatism, endocarditis, neuritis, duodinal ulcers and many other systemic disturbances. Why can't it cause thyrotoxicosis? Rosenow has shown that an infection at the root of the teeth has caused a stone in the kidney. Why can't an infected tonsil crypt cause thyrotoxicosis?

The thyroid gland is a manufacturing plant and a storehouse of colloidal material, which is iodin connected to protein, forming a compound called thyroglobulin, the active principle of which is thyroxin. This colloidal material is taken out of the storehouse by

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the lymphatics and circulation and carried to the part of the body where it is needed most; for example, to the nervous system, the vascular system, skin, hair, sexual organs, the osseous system, etc. If there is too much or too little colloidal material manufactured by the gland there will be too much or too little stored in the storehouse, therefore there will be too much or too little absorbed and carried into the system, thus there will be alterations in the aforesaid structures either as an over-stimulation or under-stimulation. It matters not what stimulates the thyroid gland to an over-production of colloidal material, it causes a thyrotoxicosis, and if we have an under-stimulation we have cretinism in the child or myxedema in the adult. The report of the following case will support my contentions:

Mrs. D., age 45 years, was sent to me Oct. 24, 1924. She was very nervous, tremors of the hands, pulse 150, eyes somewhat protruding, palpebral fissure enlarged, ankles swollen. She had been in bed five weeks because of her rapid pulse. About one year ago she began to complain of lack of endurance and rapid heart. Was east and visited a great deal, noticed hands weak and trembled, lost weight from 153 to 125. Blood pressure 150.

Examination shows both lobes of thyroid large, tonsils buried and infected, blood count: red, 4,368,000; white, 6,000. Coagulation time, 6 minutes. Urine, straw, acid, 1012, no sugar or albumin, phosphates, many epithelial cells, small calcium oxalate crystals.

After giving her calcium lactate and consulting her physician as to the condition of her heart, we gave her ether and removed her tonsils by dissection and snare. The right was very large and buried deeply, the left was full of detritis. There was so much in it that we used the sucker to remove it to prevent her from inhaling it. The right bled rather freely, enough that we used a compress for fifteen minutes, after that no bleeding.

Laboratory report on tonsils showed moderate amount of inflammatory infection and fibrotic change. The crypts showed large deposits which have undergone hyalinization and, in places, calcification. The changes represent chronic infection of crypts and some diffuse chronic inflammation.

She remained in the hospital five days, pulse 160 down to 134. Mar. 15, 1925, she was feeling fine, could hardly detect any enlargement of the thyroid gland, no tremors, no Grafe, pulse 80, blood pressure 120-78, weight 144½.

In 1913, Clement F. Thiesen, of Albany, N. Y., published a paper entitled, "Acute Thyroiditis as a Complication of Acute Tonsil-

litis". He was one of the first to point out that acute tonsillitis was the cause of acute thyroiditis which, if repeated a number of times, will in some cases at least cause goiter.

All of Thiesen's patients were young women. A short summary of his cases are as follows:

Case 1: Age 20 years. Sore throat for several days before examination which revealed a typical follicular tonsillitis. Thyroid enlarged and tender on palpation, the hypertrophy increasing during the course of tonsillitis. The swelling of the thyroid had begun during the third day of "sore throat", and the patient stated that the gland was not enlarged before the attack of tonsillitis. Under the usual treatment of tonsillitis, with an ice-coil around the neck, the attack subsided in about one week. Tonsillectomy was refused, and the next winter the patient again presented herself with a similar attack, "running the same course, and again developing with an acute tonsillitis. This patient came to the clinic at regular intervals during the next two years, and while there was no further attack of acute thyroiditis, she developed a gradually increasing diffuse goiter. It is at least possible that etiologically there is a connection between her attack of thyroiditis and the subsequent hypertrophy of the gland. There is no doubt that the infection of the gland was each time caused by the acute tonsillitis."

Case 2: Age 22 years. Same history as preceding case. Patient was practically well in ten days, and was not seen again for about two months, when she came to the clinic with typical symptoms of hyperthyroidism.

Cases 3 and 4: Age 21 and 24 years. Both stated that before the present attack they had had no enlargement of the thyroid. One patient was just getting over a severe attack of acute tonsillitis, and the other was still having an acute attack.

Case 6: Age 19 years. Very severe acute tonsillitis, which was followed by the development of acute thyroiditis. The attack ran the usual course and a year later another acute thyroiditis came on with an acute follicular tonsillitis. This patient has been under observation continuously. and has developed a well marked diffuse goiter, which started about six months after her last attack of thyroiditis.

Case 7: Age 30 years. A very severe acute thyroiditis came on directly after an acute tonsillitis. "There was a good deal of dyspnea and dysphagia in this case, and within a few months after the attack she developed a typical condition of hyperthyroidism."

It seems reasonable to suppose that recurring attacks of tonsillitis might involve a thyroid, and eventually set up a chronic condition of hyperthyroidism, even if the thyroid symptoms were never sufficiently acute to be differentiated during the tonsillitis attack.

Louis E. Brown, of Akron, Ohio, has probably done more work on the relation of tonsilar infection and thyrotoxicosis than anyone else. He not only studied his own patients but prepared a questionnaire which he sent to twenty-eight physicians. These included internists, surgeons, and ear, nose and throat men, the idea being to get a broad range of opinions. The questionnaire was as follows:

1. How many cases of goiter have you had in your practice, in which according to the patient's history, the symptoms accompanying the goiter were apparently temporarily increased during the attack of tonsillitis?

He received thirteen positive replies from such men as David Marine, Plummer, of Mayo Clinic, and Allan Graham.

2. In how many cases, were adults presented in early stages of goiter, was the growth of the goiter apparently arrested by the removal of the tonsils?

All the replies to the question were affirmative. They came from Crile, H. G. Sloan, of Cleveland, Albert J. Ochsner, O. P. Kimball and such men.

3. How many cases after thyroidectomy, in which the symptoms persisted were relieved by tonsillectomy?

Dr. J. A. Stucky, of Lexington, Ky., had seen seven such cases.

4. How many cases of young girls at puberty, who have a beginning simple goiter, had their tonsils removed in early childhood, or at least before the symptoms of goiter were evident?

Most of the answers to this "fell down", "No data" was the reply.

5. In those who have had their tonsils removed, how many were benefitted or cured by tonsillectomy?

All who answered this believed by removing an infected tonsil in an adolescent girl who had a goiter would have a beneficial effect on the thyroid condition.

Dr. Brown said, a number of patients where a thyroidectomy did not relieve the toxic goiter symptoms were not relieved until infected tonsils had been removed. A few cases seen where a tonsillectomy had been done and a small piece of infected tonsil was left the toxic goiter symptoms remained until this infected tissue was removed.

Dr. G. B. Wood, of Philadelphia, recorded a case of a trained nurse, who had recurring tonsillitis followed by exophthalmic goiter

and hyperthyroidism. After removal of the tonsils the attacks stopped for six months, the goiter began to decrease in size and the exophthalmos to disappear. Then followed a sore throat and symptoms of hyperthyroidism. At this time it was found that a small piece of the tonsil had been left. That was removed and all the symptoms of hyperthyroidism disappeared.

Shambaugh, of Chicago, under whom I have studied, warned us to look out for chronic buried abscesses where the patient does not complain of "sore throat". I have taken out tonsils where the abscess occupied half of the base of the tonsil. Many times in children I have found so much detritis buried that I would stop operating long enough to suck this poisonous material out of the throat pulmonary abscess.

Dr. Joseph Beck, of Chicago, in whose clinic I have worked, believes it much better to do a tonsillectomy than to ligate the superior thyroid artery as a preliminary step to thyroidectomy.

Dr. S. P. Beeber, of New York, after examining 3500 cases of thyrotoxicosis, says that 35 to 40 per cent of patients between 16 to 24 who have hyperthyroidism give a history of repeated attacks of tonsillitis.

Many patients with Basedow's disease with their early headaches for fear the patient would inhale it into the lungs and cause a come to the ear, nose and throat man first. It is for him to exclude eye troubles and sinusitis. Then, after examining the tonsils and larynx, he should always examine the thyroid.

Men doing thyroid work should always examine the tonsils.

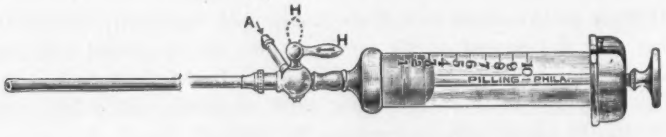
The object of this paper is to stimulate interest in this subject and if need be to invite criticism and opposition which will arouse more widespread consideration of the entire subject.

BRONCHOSCOPIC SYRINGE.*

DR. ROBERT M. LUKENS, Philadelphia.

In bronchoscopic treatment of disease of the lung and also in experimental work upon animals it is necessary at times to inject fluids into the bronchi by means of a syringe with a long cannula. Necessarily a certain amount of the fluid will remain in the cannula after the plunger of the syringe is pushed home. When large quantities of fluid are being used comparative accuracy is not necessary and the retained amount is of no moment; but where small amounts, such as graded doses in experiments upon animals, requiring cannulae of 60 c.m. lengths or longer, the quantity remaining in the cannula renders accuracy uncertain.

This can be overcome in two ways. First, by dilution, *e. g.*, increasing the bulk of the fluid; second, by blowing the retained fluid out of the cannula into the bronchus.



Dilution is not practical because of ease with which the increased amount of fluid can be expelled from the bronchus by cough.

The difficulty in blowing the smaller quantities out of the cannula into the bronchus lies in removing the syringe in order to fill it (the syringe) with air. Often when the syringe is removed the patient or the animal, as the case may be, coughs and blows the solution back into the air.

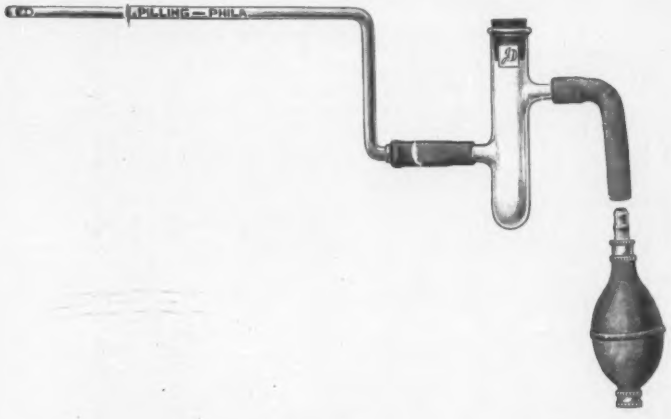
To obviate this I have placed a two-way valve with a quarter turn at the syringe end of the cannula to retain the fluid in the cannula by closing the entrance and opening an air intake hole while the syringe plunger is being pulled back. The air intake hole is then closed and the cannula opened by turning the valve to its original position and the air forced through, carrying the retained fluid into the bronchus.

*Read before the Philadelphia Laryngological Society, Oct. 16, 1924.

BRONCHOSCOPIC SPECIMEN COLLECTOR.*

Dr. Robert M. Lukens, Philadelphia.

This is an instrument for collecting specimens of secretion or pus from the bronchi in diseases of the lung. It is simply a test tube, with an intake arm and exhaust arm, which is interposed between a bronchoscopic suction tube and a suction pump. The collector intercepts the pus or secretion on its journey from the bronchus to the



suction pump. The collector is then removed with the short rubber tubing attached. The free end of the rubber tubing is then slipped over the opposite arm of the collector, thereby sealing it and the specimen is ready for transportation to the laboratory for bacteriological study and the preparation of vaccine.

The whole apparatus is sterilized by boiling. Where there is only a minute quantity of secretion or pus, this collector cannot be used because of its size. In these cases the Clerf collector is to be used.

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*Read before the Philadelphia Laryngological Society, Oct. 16, 1924.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

May 8, 1925.

Paper: **A New Artificial Ear Drum.** Dr. Harold J. Isaacs (by invitation).

Meningitis Complicating Acute Mastoiditis in a Diabetic. Dr. William Siegmeister.

Mrs. S. G., age 50 years, came to the writer's office on Aug. 6, 1924, complaining of pain in right ear for past two weeks, following cold in head. Past history: diabetes for six years, having been under doctor's care all the time; for past six months has been sugar free. Satus presence: rather obese, good color, apparently in good health.

Ear examination: left ear negative; right ear drum membrane red, bulging, all landmarks gone, slight tenderness over mastoid tip, hearing much impaired.

Diagnosis: O.M.P.A. Paricentosis done, free pus found. Patient has been coming to the office for treatment for two weeks, during which time she felt comfortable, pulse and temperature (taken every three hours) normal, except for moderate pain in the ear at night and profuse discharge from the ear. Urine examination showed glucose, 0.5, and the last examination showed albumin and the presence of acetone.

Repeated radiographs showed infiltration of right mastoid but intercellular supta could be distinctly discerned.

The writer had her admitted to the hospital on Aug. 20, 1924, for operation. He did not deem it advisable to operate in the presence of glycosuria and acetonsuria, so he ordered a complete examination of twenty-four hours' specimen of urine and blood chemistry with view of putting her on a course of insulin treatments to make her sugar and acetone free to be fit for operation. On Aug. 21, midnight, patient suddenly became delirious, temperature rose to 104,° has been delirious all night; on the next morning when seen, patient was unconscious, showed a facial paralysis on the right side, rigidity of neck, lumbar puncture showed cloudy fluid, pressure normal, on examination, 5600 cells, most of which were polymorphic nuclear leucocytes. She was operated on by the writer the same day. Operative findings: Mastoid mixed type, mostly diploic, some pneumatic cells; there was free pus in region of antrum, and posterior to the sinus. Dura was exposed. Most of the cells that were there were intact. Diagnosis: Acute mastoiditis, coalescent type, which didn't coalesce completely. Patient did not rely after the operation. Expired at 2 a. m., Aug. 24.

Analyzing this case, the question comes up of the advisability of operating early in diabetics, in the presence of glycosuria and acetosuria. We are all loath to operate on diabetics unless there is an absolute indication. Taking this case, if the patient were not a diabetic the writer believes that most otologists would not have operated, with such a clinical picture and X-ray findings. The operative findings showed that we were dealing with a coalescent type of mastoiditis before coalescence took place. We usually wait with the operation until after the fifth or sixth week, to give nature a chance to localize the infection. In the presence of glycosuria and acetosuria, the writer thought there was an absolute indication for waiting, having such a valuable remedy as insulin at our command, which accomplishes the expected results in such a short time. Yet, the results proved that he was entirely wrong. Possibly, if the patient had been operated on a few days before, she might have escaped the complicating meningitis, and might have had a chance of recovery from her mastoiditis.

Acute Suppurative Labyrinthitis in Diabetic. Dr. William Siegmeister.

Mrs. R. T., age 50 years, came to my office, Mar. 8, 1925; referred by Dr. M. Clurman.

Chief complaint: Running ear for past four weeks.

Family history: Father and mother had diabetes; recently a brother died from some malady.

Past history: Had diabetes for past sixteen years; subject to frequent attacks of diarrhea.

Has ten children, all living and well. Emphatically denied any previous ear trouble.

Present illness: About four weeks ago had an attack of tonsillitis, which was followed by earache, headache and dizziness on left side. A few days after left drum membrane ruptured, followed by sanguinopurulent discharge. Pain in the ear was relieved immediately. She had been treated by her family physician since. Feeling comfortable, except for occasional headaches and dizziness.

Physical examination: Left ear: profuse discharge, large central anterior, inferior perforation of membrana tympani discharge reappearing immediately after being wiped away. Slight sagging of posterior superior canal wall, slight tenderness over mastoid. Right ear, nose and throat, negative.

Functional examination: Hearing in right ear normal. Left ear (with the use of noise apparatus) entirely gone, for voice whisper and all tuning forks. Weber was still lateralized to the left.

Neurological examination: Spontaneous rotary nystagmus to right side, otherwise negative, reflexes normal. Pupils and pupillary reactions normal.

Labyrinthine examination: Douching left ear for four minutes with hot water at 110° F. No nystagmus to left, nystagmus to right persisted, no nausea, vertigo or falling, no past pointing. Douching of right ear with cold water at 68,° nystagmus to left, falling to right. Past pointing to right, nausea being present. Fistula test negative. From above examination dianosis of acute mastoiditis, complicated by acute diffuse suppurative labyrinthitis was made and patient referred to hospital. Examination on admission: Temp. 98, pulse 84, urine SP. Gr. 1026 albumin ++, glucose 2.5, acetone, diacetic acid plus, plus. Blood examination, R.B.C. 4,000,000, Hb. 78 per cent, W.B.C. 10,200, polys, 79 per cent. Bl. sug. 250. Lumbar puncture; pressure increased 4000, cells prm. polys. 90 per cent. No organism and direct smear found.

Mar. 9: Patient comfortable, at 12 M. Temperature rose to 103.2,° 4 p. m., 104.° She was operated on at 5 p. m.

Operative findings: Local anesthesia, radical mastoid done. Entire body of mastoid found necrotic, filled with pus. Sinus found exposed, dura of middle fossa exposed. No labyrinthine fistula found. Condition after operation did not improve. Temperature persisted. Two days after she developed convulsions. Exitus on Mar. 14, 1925. A part of the labyrinth was removed. Post-mortem. The pathological report is as follows: Sections of this tissue show varying stages of inflammation. Some areas show an acute exudative inflammation characterized by moderate absorption of the bone tissue with invasion of all types of inflammatory cells. Other sections show presence of granulation tissue composed of collagenous fibrils, young fibroblasts, small round plasma and polynuclear cells. This tissue is non-specific in character. This case shows following points of interest:

1. Acute, suppurative, diffuse labyrinthitis occurring in an acute otitis.

2. Patient suffering from acute, diffuse, suppurative labyrinthitis without any classical symptoms. There was no nausea, vomiting, vertigo or atasia.

3. Question of labyrinthectomy is up for discussion, there being all the signs of a dead labyrinth with threatening meningitis, it was my opinion that the labyrinth should have been opened or exenterated.

Dr. Kopetzky did not agree with me. I would like to hear your opinion on the question.

DISCUSSION.

DR. M. L. HARRIS said that in the case reported by Dr. A. A. Schwartz he had been the first to see the patient, who came to him with an abscess of the septum, which he incised; three days later she developed an otitis media, and four days after that was admitted to the hospital.

Paper: **A Report of Three Cases of Primary Acute Mastoiditis.** Dr. Samuel J. Kopetzky and Dr. Ralph Almour.

Paper: **Two Cases of Primary Jugular Bulb Thrombosis, Not in Infants.** Dr. William Ginsberg.

Acute Mastoiditis with Complicating Facial Paralysis; Recovery After Operation. Dr. A. Greenstein.

This case is not reported on account of its rarity, but rather to bring home to the otologist once more the fact that facial paralysis occurs as a complication in acute mastoiditis.

There is no need, at this time, to go into the details of the causes of facial paralysis; suffice it to say that it can occur due to extension of inflammation along the course of the facial nerve, whether in the facial canal, in the internal auditory meatus or as a result of bone destruction within the mastoid itself.

The degree of facial paralysis varies, depending upon the extent of the inflammation, whether the entire or only certain bundles in the nerve are affected.

While the paralysis may be caused by lesions of the nerve located centrally, during its course through the Fallopian canal, or peripherally, it is desired in this report to consider it only in its connection with an acute mastoiditis.

D. C., age 9 years, two days after surf bathing, on July 30, 1924, patient complained of pain in right ear, slight cough and fever; after several hot ear irrigations the pain subsided.

A week later, on Aug. 7, 1924, I was called in to see the patient. She again complained of pain in the right ear. A right sided facial paralysis was noted, involving the upper branches of the facial nerve. There was no loss of sense of taste. Temp. 101° F.

On physical examination a bulging drum was found, dark red in color, with all the landmarks and light reflex gone. There was no headache, vertigo, or nystagmus. Myringotomy was performed.

The next day the patient still complained of pain in the ear; there was only a small amount of purulent discharge, although the tympanic membrane was widely opened. She complained at this time of some tenderness over the mastoid tip, but not over the antrum. Hearing in the right ear was somewhat reduced, but did not lateralize.

X-ray examination showed clouding of the mastoid along the posterior wall and tip.

Blood examination: white cells, 14,000, with 73 per cent polys.

Examination of the ear on this day showed some sagging of the post-superior canal wall.

In view of the fact that this patient had a facial paralysis, although the mastoid symptoms were not marked, it was deemed advisable to perform a mastoid operation.

On Sept. 9, 1924, the usual mastoid operation was performed. The cells were broken down and filled with a large amount of thick, greenish purulent material. The sinus wall was diseased and the sinus on being exposed appeared normal. Acqueductus Fallopii also appeared normal. The wound was packed and dressing applied.

A few days after the operation the facial paralysis began to show signs of improvement so that on Sept. 18, 1924, the patient was able to wrinkle her forehead and partly close the eye. The middle ear be-

came dry by the third week and the mastoid wound healed in five weeks, during which time the paralysis continually improved and when discharged had completely disappeared.

In differentiating this condition from other cause of facial paralysis, the most important one is the idiopathic Bell's palsy. In view of the fact that this paralysis began to improve immediately after the mastoid operation, it is only fair to assume that necrosis of the mastoid bone was the cause of the facial paralysis in this case.

DISCUSSION.

DR. WM. SIEGMEISTER, referring to the diabetic cases, said he had had a number of cases with middle ear infection in diabetics which have recovered, but these cases had given him a great shock. the first being only two weeks old and the patient being in such apparent good condition. She was admitted for operation only because she was a diabetic, and it was just one day too late.

In the second case, about the labyrinthitis, there was some misunderstanding in the discussion. It was not a case of labyrinth examination. Examination of the labyrinth showed an absolutely dead labyrinth; it did not respond to any of the tests; there was no hearing whatever. Dr. Kopetzky was in doubt as to whether the infection in the labyrinth was from the middle ear, or from meningitis, and he thought she had meningitis, from the spinal fluid report. When the patient was first examined by Dr. Siegmeister she lateralized to the left, showing that the labyrinth had some function left; on the next day she lateralized to the right; from that it would seem that the infection traveled from without inside, and that the meningitis was secondary, etc.

Paper: **Case of Acute Mastoiditis with Meningitis. Temporosphenoidal Abscess Discovered at Post-Mortem.** Dr. J. M. Polisar.

Paper: **Temporosphenoidal Abscess and Mastoiditis of Doubtful Origin.** Dr. William Siegmeister.

DR. KOPETZKY said that the first series of cases reported, namely, those complicated by diabetes, brought home the fact that neither the general practioners nor many otologists seem to realize that the diabetic condition modifies and controls the clinical picture, and that the factors which one ordinarily looks for in the development of mastoiditis are clouded by the underlying constitutional trouble. These cases frequently run a seemingly inocuous course, until a point is reached when they present a more pronounced clinical picture. At that time, they have frequently passed beyond the stage where effective surgery can be employed; and the surgeon is face to face with intracranial complications. This is exactly what happened in the cases reported.

Dr. Kopetzky spoke of a case which he had recently operated upon. It had been under observation for over four weeks and then suddenly developed a large abscess in the neck, extending down to the clavicle. The medical attendants on that case were amazed, at operation, by the extent of the process which had developed under their very eyes, without their having appreciated what was happening.

Dr. Kopetzky said that while diabetic patients present neuritides generally, when it comes to the mastoid the constitutional trouble seems to be anesthetizing and the patients do not as a rule complain of pain, either spontaneously or upon pressure. On the whole, one cannot judge a diabetic mastoid by the criteria employed in the average case.

In the case presented by Dr. Schwartz, in addition to the unusual finding of a streptococcus mucosus capsulatus in the blood, there was the element of diabetes. The patient had been watched and studied by competent observers; and yet, as the story shows, the clinical picture was again misjudged because of the underlying constitutional disease. According to Dr. Kopetzky, the diabetic patient who has an ear infection which does not promptly resolve and show signs of healing should be operated upon.

Roentgenological examination helps greatly in these cases and should be performed repeatedly so that any progressive changes in the bone

may be noted. Destruction in the bone can be studied in this way unless there is a scrolosis of the bone present. The latter condition is readily established by comparison with the condition in the opposite ear, because usually one finds the same type of mastoid process on both sides.

It is to be stressed that Dr. Schwartz' case presented as a predominating symptom frontal headache and headache localized more or less on the affected side of the head. Headache which is fairly intense on the side on which mastoid operation has been performed is very suggestive of sinus thrombosis.

The cases presented by Dr. Almour are exceedingly interesting. The first case we studied from its very commencement, and it developed under our eyes. It was very difficult to believe that a mastoid was developing in that case, for our daily examinations of the middle ear never showed any signs of trouble which could be noted as abnormal. The case of sinus thrombosis for which we did not operate, and which occurred in a case of primary mastoiditis, was in the care of physicians other than ourselves at its inception. The report was given us that the child had had erysipelas, following which the temperature became normal, and that the mastoiditis developed subsequently. At operation, when the sinus was exposed, we found an organized clot; the vein was firm and hard and showed no pulsations. The child had a flat temperature at the time and ran none subsequently. Dr. Kopetzky thought that the child had had, not an erysipelas, but a sepsis due to a sinus thrombosis, and that the indefinite rash described by the medical attendants was really a rash due to streptococcus infection. Of course there can be no proof of this; but the subsequent course of the case would seem to verify it. Time passed, the clot was organized, and there was no systemic nor other reason to disturb it. Unfortunately, a month or so later the child died of acute nephritis, after she had been discharged from our care.

In the case of primary bulb thrombosis reported by Dr. Ginsberg, the diagnostic picture was confused in the beginning because of the suspected pulmonary condition; and the man who operated could hardly be blamed for going at it half-heartedly. He said that he found nothing on opening up. This case presented to Dr. Kopetzky's mind all the symptoms of a sinus thrombosis; but although he persisted in his diagnosis, he could not get the support of the rest of the staff on it, and the family refused permission to operate. Later, when permission was granted, the thrombosis was established to the satisfaction of everybody. Dr. Kopetzky holds that where a case presents a running ear and a sepsis with a positive blood culture not explicable by any lesion elsewhere in the body, one should always suspect a sinus thrombosis, whether the patient be an infant or an adult. No one has yet estimated the number of times in young adults where there is a bone dehiscence between the floor of the tympanum and the dome of the jugular bulb. Some years ago Dr. Kopetzky figured this roughly as happening in eight out of twenty-five specimens. In the majority of cases in a general hospital, these patients come in with secondary lesions and the sepsis due to the secondary lesion overshadows the primary focus in the lateral sinus. The case should really be treated before the secondary lesion develops.

Dr. Polisar's first case was interesting, Dr. Kopetzky said, because he had never before seen a multiple temporosphenoidal abscess accompanying a thrombosis, as here. When Dr. Kopetzky first saw the child, it was sitting up and no one could conceive that it had a brain abscess. All that Dr. Kopetzky found was a sluggish reaction to light in the right pupil and an ankle clonus; and he advised a lumbar puncture and hospitalization. It was very hard to make the child's parents agree to this in view of its apparently good condition at the time of consultation. The diagnosis was established upon lumbar puncture. Dr. Polisar had suspected a brain abscess, but Dr. Kopetzky could not agree to that diagnosis in view of the facts that he then had at his dospisition. When

the child was operated upon for drainage of the cisterna magna, it was found that the fluid did not come out with the usual gush and did not seem under great pressure. Then, for the first time, the operator was doubtful as to the condition for which he was operating. However, the child, who was operated without anesthesia, as obviously moribund; and the surgery was undertaken as a last hope. Autopsy revealed an erosion of the dura near another erosion found in the tegmen antri which had not been noted by the original operator at the original mastoidectomy. Autopsy revealed not only these erosions, but a brain abscess with a lining, delimiting membrane. In view of these findings, Dr. Kopetzky doubted whether the child could have been saved in any event; but the case demonstrates how large a collection of pus can be carried around without destroying the patient's complete control of all his faculties.

In regard to the finding of granulations on the dura in the middle cranial fossa at an original mastoidectomy, the question arises as to whether they should or should not be disturbed, whether the operator should consider them as a protective barrier and leave them alone, or search through them for possible fistulae leading intradurally. Dr. Kopetzky holds the following. If an extradural abscess is found at original mastoidectomy in a patient who presents no signs nor symptoms of an intracranial nature, neither headaches nor focal signs, then granulations found on the dura of the middle cranial fossa should be left intact. If, on the other hand, the patient shows signs of meningeal irritation,—localized headache or focal signs due to pressure—then one is justified in suspecting that these granulations are but the outer covering of an intradural or brain abscess, and one should not leave them intact. Had the case which Dr. Siegmeister reported, and which presented granulations in the middle cranial fossa in conjunction with intracranial symptoms, been probed, the intradural and brain abscess would have been uncovered. In view of the grave condition of the patient, it is doubtful whether this would have affected the outcome of the case; but at any rate a full diagnosis *intra vitam* would have been made.

Finally, in the question which was raised by Dr. Siegmeister as to whether or not one should remove a dead labyrinth in a case of meningitis, Dr. Kopetzky holds that it is better to drain the meninges than to prolong the procedure and operate on the labyrinth first.

Dr. Chambers said that when he looked at the menu of entertainment for the evening he was impressed by the fact that eight speakers were on the list "by invitation". He recalled the time when there was no Section on Otology, and he felt that this was the first time in the history of the Section when such a rich programme was presented from the outside. So rich a programme of such beautiful cases so beautifully presented was a great treat, and Dr. Kopetzky was to be congratulated on his staff; it must be wonderful to have such a class of boys.

In speaking of his artificial drum, Dr. Isaacs had stated that it required the use of the speculum, to place the cotton properly, to stop the tinnitus and relieve the deafness. Dr. Chambers said that he had had many patients who were in the habit of putting a little ball of cotton in the ear themselves, and he had found that they could do it better than he. They never used anything but dry cotton, and it seemed to serve for a long time.

Dr. Maybaum said that only words of commendation should be given the men who presented these interesting and instructive cases of otitic complications. As he listened to the reports, he could only conclude that the trials and tribulations of an otologist in one institute resemble very much those of men in other institutions.

The question of operative interference in cases of acute mastoiditis complicated by an acute suppurative labyrinthitis is one of the difficult problems which occasionally presents itself for decision to the otologist. In general he believed that operative interference upon the mastoid should be delayed as long as possible. Repeated examination

of the spinal fluid, close observation of the course of the disease, will aid in determining the question for or against a labyrinthectomy.

The case of sinus thrombosis reported by Dr. Schwartz is extremely interesting. He not only had a diabetis to deal with, but also a streptococcus mucosus infection. Dr. Schwartz referred to the symptom of early headache in this case of sinus thrombosis. Dr. Maybaum does not believe that headache is a common symptom of an uncomplicated sinus thrombosis. In the case reported by Dr. Schwartz, the characteristic headache of a Gasserian ganglion involvement seemed to be a prominent symptom; such headaches he had seen in quite a few cases of mucosus infection involving the petrous portion of the temporal with extension to the Gasserian ganglion.

As for the three cases of primary acute mastoiditis presented by Dr. William Ginsberg, even though the history of the onset of these cases may be very vague or apparently absent, something of an inflammatory nature had taken place in the middle ear. Careful inspection of the tympanic membrane usually discloses some change of a more or less minor nature. The drums are not absolutely normal. Some thickening or dulling of the landmarks may be the only signs of previous middle ear in volvement. Nothing was said in the report of these three cases of so-called primary mastoiditis, as to the type of infection cultured from the mastoids.

The cases of primary jugular bulb thrombosis presented by Dr. Ginsberg are worthy of comment. The diagnosis of primary jugular thrombosis can be one of the most difficult in otology, unless the signs and symptoms are quite definite. One should not be too hasty in making this diagnosis; having made such a diagnosis, definite action must necessarily be taken. The presence of a discharging middle ear without mastoid involvement, a streptococcic bacteremia, early ocular fundus changes—papillitis—enlarged tender glands at the angle of the jaw point quite definitely to a primary jugular bulb thrombosis. In some of these cases of primary jugular bulb thrombosis which we observed in Dr. Friesner's service at Mt. Sinai Hospital the ear had never discharged.

Dr. Maybaum believed that clinical meetings such as those should be held with even greater frequency, as the benefit resulting from the exchange of views and experiences is invaluable.

DR. GINSBERG: Dr. Maybaum understood me to say that a choked disc was present on admission and that a diagnosis of primary jugular thrombosis could have been made therefrom. I corrected him by saying that there was no evidence of any papillitis or choked disc on admission; that the choked disc did not appear until four weeks after admission and persisted for six weeks, after which it began to recede.

DR. LEDERMAN said that in diabetics, unless the indications were exceedingly urgent, surgery should be held in abeyance. He is inclined to look upon diabetes involving the ear with a great deal of trepidation and feels that unless one can be reasonably sure that the subjective symptoms are due to the collection of pus, it is good judgment to watch such cases and treat them symptomatically. Simply because a diabetic has an otitis, with no pain, is not an indication for going into the mastoid. Six weeks ago, the mother of a physician developed an otitis following influenza. When first seen there was no question of draining the middle ear, as there was decided bulging of the drums and tenderness over the mastoid tip and antrum. She did very well for a few days and was given insulin in anticipation of the possibility of surgery. The objective symptoms were not very satisfactory, the swelling of the membrana tympani did not go down very promptly and drainage was very profuse. The insulin was continued, however, and simple suction was employed in a gentle manner, plus the dry treatment with iodin powder, and in that case although sugar was present and disappeared under the insulin treatment, it returned when same was omitted. The mastoid tenderness did not disappear very rapidly and the secretion continued for four weeks, with the prominence of the ear

drum. It required fortitude to practice masterly inactivity, but the condition subsided without surgery. That was an illustration that when a patient has diabetes and rather suggestive clinical symptoms of mastoid disease, it is not necessarily an indication for operation. Such cases are very poor surgical risks.

Dr. Lederman then told of another case, a woman age 56 years who had been under the care of a colleague who was taken ill. He had been asked to take charge of the case for the time, and did not think it was one which required immediate operation, although he felt that the mastoid was involved, but the ear was draining and there were no retention symptoms. He suggested that another colleague see the case and this man advised against operation; but a third man who was called in urged operation and his advice was followed and he operated, and the patient promptly developed coma and died.

Dr. Lederman said he had seen other cases of diabetics with suppuration of the nasal sinuses that got well under palliative treatment, and he could not be too emphatic about operating on these patients without proper consideration of all factors.

He did not recall seeing an acute mastoiditis without some middle ear involvement, and he did not know of any case where he would say it was an acute mastoiditis *per se*. He agreed with Dr. Friesner and others that there had been some previous infection with an occlusion of the aditus ad antrum.

Referring to kidney involvement associated with acute conditions of the middle ear, Dr. Lederman said he had seen a number of cases where the kidney condition was present at the time of a middle ear involvement and when the middle ear improved the kidney condition improved also, showing that undoubtedly there was some connection. He also agreed with Dr. Maybaum that in the presence of an acute mastoiditis with labyrinth symptoms, he would not be tempted to open the labyrinth at the mastoid operation, and said that he had seen a number of cases where the labyrinth condition improved after the mastoid had been exenterated.

He expressed regret that he had not arrived in time to hear Dr. Isaacs' description of his artificial drum, and said that he himself had used pieces of rubber tissue cut large enough to rest on the remnants of the drum, allowing them to remain in place two to three weeks or longer, depending upon the local condition, though they sometimes cause a little maceration of epithelial tissue and must then be removed. He believed the drum recommended by Dr. Isaacs could be introduced more easily and would be less irritating.

Dr. Ralph Almour, closing the discussion, stated that the principle of affording drainage to a suppurative focus is one of the fundamentals of surgery, and that this principle is in no way altered by the presence of diabetes in an individual. While it is true that diabetic patients react poorly to surgery, it is also true that they offer no resistance to offset or localize an infection. Consequently the infection in the mastoid process presents a danger far greater to life than the operative measures to relieve it, and where the operation is performed under local anesthesia and proper measures have been instituted to combat acidosis, the patient's condition has been improved by the surgery on the mastoid. He also feels that it is unwise to render a chronic diabetic entirely sugar-free before operation and that the risk is much less, as regards acidosis, when the patient is operated upon in the presence of a small quantity of sugar in the blood.

He agreed with the previous speakers that in the cases of primary mastoiditis which he had presented there must undoubtedly have been some middle ear infection at one time or another. What he meant to impress upon the listeners was that none of the cases which he had seen had presented any clinical or otoscopic picture of middle ear disease. The drums in each of the cases were normal in appearance. In answer to Dr. Maybaum, he regretted that no bacteriological studies had been made.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Meeting of February, 1925.

The Importance of a Laryngeal Examination in Adults Prior to an Operation for the Removal of Tonsils. Dr. Louis Baer.

While the laryngologist will readily advise the removal of tonsils, when indications for their removal, local or constitutional, are present, he is not at all unmindful of certain complications and dangers that may attend or follow the operation. With all our present knowledge of the anatomical structure of the tonsils, and their anatomical relation to the surrounding organs; and having at our disposal the most modern instruments and appliances to make the operation more thorough and complete, and to obtain a clean operating field, we are still fearing some unexpected complication that may occur during or after the operation. Indeed, tonsillectomy should be regarded as a difficult surgical procedure and classed among the major operations rather than minor surgery.

Complications following tonsillectomy are the exception and in trained hands their percentage is negligible, yet accidents do occur among the best surgeons. Now and then we hear of a fatality from an uncontrollable hemorrhage, or from a local anesthesia. Otitis media, pneumonia and pulmonary abscess, in their order of frequency, are complications often reported in literature. A patient of mine, a boy, age 8 years, is now under the service of Dr. C. Jackson at the Jefferson Hospital, being treated for laryngitis following tonsillectomy performed three years ago by one of the most eminent surgeons in Chicago.

Tuberculosis of the larynx is another complication, which as we have observed, was influenced by operation on the tonsils. Whether the tonsillectomy was the direct cause of infection of the larynx, as statistics gathered by Mayo and Rarabertson show that 8 per cent of tonsils removed are tuberculous. That during the operation the infected blood, serum, or pus from the tonsils either by directly coming in contact with larynx or carried by the lymphatic channels that become exposed as a result of the operation, to the lungs and from there to the larynx. Or the tonsillectomy, by temporarily reducing the vitality of the individual, flared up an already infected larynx, I am not prepared to tell.

However, two cases came recently under the care of the nose and throat service of the Mt. Sinai Hospital with an unquestionable diagnosis of tuberculosis of the larynx in an advanced stage. Both cases gave definite histories that their trouble in the throat started immediately following tonsillectomy. As the time elapsed between the operation and the diagnosis of tuberculosis of the larynx was a few weeks, I am inclined to believe that in both the larynx was diseased before the tonsillectomy was performed.

Case 1: J. A., age 40 years, came to the Mt. Sinai Nose and Throat Clinic, June 2, 1923, complaining of sore throat. After a thorough nose and throat examination, the following diagnosis was recorded on his card : Chronic sinusitis, chronic hypertrophic rhinitis, chronic pharyngitis, diseased and hypertrophied tonsils, and chronic laryngitis. There was no evidence at this examination of the larynx to arouse a suspicion of tuberculous infection. The chronic laryngitis found was thought to fit in the picture of a generalized chronicly inflamed nose and throat, possibly due to the sinusitis or to the diseased tonsils. He received local treatment and was advised to have his tonsils removed.

Nov. 6, 1923, tonsillectomy under local anesthesia was performed by the writer. He was not examined, except the resident physician who is taking charge of admission of patients, since June 2, 1923.

A few days after the operation he came to the clinic complaining of a sore throat. An examination of the throat showed the ordinary post-operative picture. There was no evidence of injury to the surrounding structures. The larynx was not examined because nothing extraordinary was suspected. A gargle of aspirin, 10 grs. in one-half glass of water, was prescribed (t.i.d.).

He returned within a few days reporting that he felt better, but his throat is still sore, and was advised to continue the same gargle.

In February, 1924, about three months after the operation, he came to the clinic complaining again of a sore throat. He stated that since the operation the pain in the throat never left him completely, but persisted with moderate severity, for which he doctored himself with the aspirin gargle. But as he felt that he is getting worse he came to be examined. After a complete examination, we were astonished to find Koch's infection of the larynx in an advanced stage. The epiglottis was swollen, edematous, more than half-an-inch in thickness, with ulceration on the anterior surface near the upper margin. The arytenoids were swollen, the ventricular bands markedly thickened, obstructing from view the vocal cords, which could not be seen.

Wassermann test was negative. Sputum examination showed T.B.

Examination of the lungs showed definite tuberculous involvement of both lungs. He was treated for a while at the Mt. Sinai and Phipps Clinics, and in May, 1924, was sent to the Hamburg, Pa., Sanitarium. Left the sanitarium in October, and in Philadelphia was wandering from one clinic to another. His epiglottis, when I saw him last, was almost entirely gone, and the entire larynx is in a state of ulceration. Died Dec. 4, 1924.

Case 2: W. H., age 29 years, came to the Jefferson Hospital in January, 1924, complaining of a sore throat. He was advised to have his tonsils removed. April, 1924, his tonsils were removed in the Jefferson Hospital. Immediately following the operation he began to feel pain deep in the throat, for which he was treated at the nose and throat clinic of the hospital. About two weeks after the operation, on the advice of his family physician, he came to the Mt. Sinai Hospital Nose and Throat Clinic.

On examination the following was found:

Nose: Some congestion of the mucosa of both chambers, otherwise negative.

Throat: Tonsils successfully removed, anterior and posterior pillars not injured.

Larynx: Epiglottis markedly swollen, edematous and ulcerated on the anterior and posterior surfaces. Arytenoids swollen. Right arytenoid ulcerated. Ventricular bands swollen. Vocal cords congested.

Wassermann test was negative.

Lung examination showed consolidated areas in both apices. Sputum examination showed the presence of Koch's bicillus.

June 26, 1924, was admitted to the Rush Hospital, but left the hospital July 5, 1924, to be admitted two days later in the Eaglesville Sanitarium, where he died on Sept. 16, 1924.

In both cases the patients had no pain in the throat prior to the operation. Their first complaint of sore throat that brought them to the hospital was due to an attack of tonsillitis. They were treated accordingly and were free from pain until after the operation.

There is no doubt that the larynx in both cases had pathology before the tonsillectomy was performed, especially in Case 2, where only two weeks after the operation there was marked involvement with ulceration of the epiglottis and arytenoids. In Case 1 the patient had constant pain in the throat for three-and-a-half months, starting immediately after the operation the tonsils until the day when the diagnosis of tuberculosis of the larynx was made, when it was found to be in an advanced stage.

I believe both cases illustrate the ill effect of tonsillectomy on a diseased larynx of Koch's infection. They also indicate the vital impor-

tance of determining the condition of the larynx, even in an apparently healthy individual, before undertaking the operation ofr removal of the tonsils.

Dr. Baer in closing: Both cases were operated under local anesthesia. I believe every patient's chest should be examined before operation, whether it is a ward or a private patient. These two cases in my opinion show that an operation flared up a tubercular condition.

Just as an illustration, I had a patient who was apparently a healthy man. He met with an accident and on the following day became hoarse. An examination showed that his throat was markedly involved with ulcerations of the epiglottis and arytenoids and died of laryngeal tuberculosis five weeks after the accident, showing that the accident flared up the condition.

DISCUSSION.

DR. M. S. ERSNER: I believe Dr. Baer has touched upon a most vital subject. Papers like these remind one of the precautions we are to take before attempting a tonsil operation. We are usually concerned with ether complications, pulmonary abscesses and hemorrhage, but are prone to forget constitutional diseases. We only realize it after we have stumbled into pitfalls.

To reiterate, it is up to us to take all precautions possible and by that means only, can we avoid unforeseen complications.

DR. STOUT: I think you can usually determine whether patient has tuberculosis or not before operation. Generally everything that happens after an operation is attributed to the operation. I do not think that the operation was the cause of these two cases mentioned by Dr. Baer.

DR. ZACKS: I believe that starvation plays an important part in these cases after the tonsils are removed because the patient is unable to get the proper nourishment owing to the post-operative discomfort. They must be regarded as potential tuberculous patients and the border line which represents their health margin is very narrow. The operative trauma, discomfort, and period of starvation which follows, which may be from one to ten days, may be sufficient cause to do the damage. This type of patient requires a great deal of food to counteract the general systemic condition.

DR. OLSHO: I would never remove tonsils in an adult unless the patient has previously been examined by an internist. At the Phipps Institute the tonsils of tubercular patients are not removed.

DR. LAESSLE: In Dr. Baer's cases, I would like to know if he has been using local or general anesthesia. The consensus of opinion, as I understand it, is that tuberculosis of the larynx is secondary to pulmonary, and we know that we might light up a dormant stage of tuberculosis when we administer general anesthesia.

The Postural and Non-Surgical Treatment of Empyema of the Antrum. Two Cases and a Novel Posture. Dr. Sidney L. Olsho.

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Dr. Olsho points out the importance of postural measures in the treatment of antral infections either with, or independently of surgery. He draws attention to the advantages of postural treatment particularly in children, when it is desirable to avoid surgical procedures. In the treatment of the latter a novel posture is demonstrated.

A part of the office antrum irrigation treatments, after shrinking, are made with the patient in Escat's position, that is head far down and opposite ear up, and followed by the expulsion of all fluid from the antrum by air before the canula or Lichtwitch needle is withdrawn, the patient meanwhile remaining in the same position.

In using suction, strong negative pressure is condemned. In preference to a machine he recommends a BD Co. ½ oz. Asepto Urethral Syringe consisting of an all-glass tube which has a bluntly tapering end which fits the nares and a thick rubber ball which after compression exerts ample pull.

If used for effect upon the antrum, one should take advantage of the force of gravity by employing it with the patient in the position mentioned, head far down and opposite ear up. Nose blowing is one of the best forms of suction, and this should be done while the head is lowered. This position for noseblowing favors the evacuation of the antrum, also when a permanent opening into the inferior meatus has been surgically established, as the floor of the antrum is lower than the floor of the nose.

He cites a case, G. F. W., age 44 years, who had a long standing, undiagnosed antral infection of dental origin, whose prompt recovery is attributed: First, to the removal of the offending tooth. Second, to the office treatments, which included antral irrigations and suction carried out to a considerable extent with the patient in Escat's position. Third, this patient is in the habit of going through certain exercises daily which bring his head near the floor, which therefore favors the evacuation of the antrum. He is directed to use a well known shrinking oil, fifteen minutes before taking these exercises. This oil contains: cocain alkaloid, camphor, oil of cinnamon, liquid petrolatum.??? Fourth, he is directed to lie on his abdomen, flat on his bed each morning and night with his head hanging over one side toward the floor, so that he can see under the bed. He is directed to instil the oil high in his nose about fifteen minutes before. He is to blow his nose well while his head is in this lowered position.

In the second case cited, a boy age 5 years, had a tonsil and adenoid operation one year previously which had not had a favorable influence upon the constant, thick, greenish nasal discharge, of which he complained. Both antra were dark on transillumination, and bilateral antral empyema was confirmed by the X-ray.

In the successful treatment of this patient, a novel posture is demonstrated (illustration). No surgical procedure, not even a diagnostic antral puncture was employed. The office treatments every three or four days consisted simply of shrinking the turbinates, followed by a very slight irrigation of the nose, then after a lapse of fifteen minutes the patient was held by his feet, with head downward and asked while in this position to blow his nose well. Suction was applied by means of the urethral syringe, with patient in the same position. Subsequently, sometimes the nose was again slightly irrigated with warm salin solution, and the treatment finished with an oil nebulization.

The home treatment was similar. The nasal oil was instilled, and one of the parents held the boy by his feet, head downward, as shown in the illustration. The patient was asked to blow his nose very well. Improvement was very marked after a short time. The patient was discharged when the transilluminations were finally persistently negative, and all nasal discharge had disappeared. The postural measures at home were continued uninterruptedly throughout the course of his treatment.

Dr. Olsho closing: Some questions that were asked have been answered by other men. I wish to emphasize that posture is a valuable accessory to any form of treatment.

Air should not be injected into the antrum before irrigating. Beck advises suction, to determine whether the needle point is properly oriented.

DISCUSSION.

Dr. Hitschler: So much is said concerning needle puncture of the maxillary sinus that we sometimes forget we can lavage the sinus via the natural opening, or via an accessory opening when the latter is present. This procedure is successful in about 20 to 25 per cent of cases; in the sphenoid in about 30 to 40 per cent. For this purpose a very delicate hard rubber canula is used. It may be easily curved to suit any particular case. The rubber canula causes no discomfort, especially when slight anesthesia is established, and is much less objectionable to the patient than the needle puncture.

Dr. Hunter: I think the rubber bulb mentioned would be harmful if used in the ear as suggested in the discussion. If a bulb is to be

used by the patient it should be tested with a vacuum gauge to find out the amount of suction it will give. I find that some bulbs give 12 to 15 inches of vacuum. Suction should always be controlled by an accurate gauge. Two inches are sufficient for the ear and 6 to 10 for the nose. No machine pump should be used without a gauge and by pass attached.

DR. P. STOUT: Cameron lights are very good not only for antra but for mastoids. They have a regular mastoid light. In asthmatic cases, even if the antra are cloudy on transillumination and in the X-rays the washings many times are clear.

When I was a youngster, people used to use snuff. I think they must have derived some benefit from it because it caused them to sneeze, but it was a bad habit to form.

DR. H. B. COHEN: I think Dr. Olsho has given us a very good paper followed by an excellent discussion. I thank him for the opportunity of listening to it.

We have to vary our treatment of maxillary sinusitis according to our patients, putting the patient on his abdomen, head hanging down over the bed and inserting drops we have used for a number of years so that the drops would reach the upper sinuses. The osteum, in my opinion, is not a true window, but canal-like, whose direction is up, back and in or down, forward and outward. Placing the patient in such a position is described by Dr. Olsho and is similar to the one we have been directing our patients at Medico Chi and in private practice, will direct the discharge towards the osteum, but I cannot see how a large cavity with but one opening can expel the heavy mucoid discharge seen in acute cases. It requires another opening for air inlet and that we make with the Tichwartz needle and aid it by the pressure of air and water, either one or both. The position mentioned may be helpful in children and occasionally in adults and I think that is what Dr. Olsho wishes to convey. We cannot treat all cases alike. In the first stage of acute maxillary sinusitis with its inflamed membranes I think one can wait a day or two for the "reaction of inflammation" before puncturing the antrum when it will not be so painful as the needle punctures the inner antral membrane. I cannot see where this position can be used other than in chronic cases. The postural position is not as good as the needle. Blowing the nose only blows out what is loosened and has overflown in the middle nasal passage. Acute maxillary sinusitis is not treated immediately with the needle, but as I have mentioned above.

DR. S. SKILLERN: We all know that the maxillary ostium, being placed in the antero-superior portion of the sinus at the junction of the superior and internal walls, is in the most unfavorable position for drainage. It was in a favorable position when we walked as apes, on all fours, but as soon as we assumed a more or less erect posture, it changed to the most unfavorable site, consequently the only time the maxillary sinus drains is when we are lying down, or have our heads on a level with, or lower than our shoulders and the moment our purulent maxillary sinus cases raise their heads, drainage ceases and the sinus starts to fill. Dr. Olsho, in standing his cases on their heads or having them lie flat on the bed and look under the bed for their shoes is putting the case in the correct position for drainage. But lying flat on the bed without a pillow on the unaffected side, thus having the affected side up, will give just as good drainage.

In my opinion, the X-ray is an excellent adjunct, not as good as transillumination, but useful in making diagnosis. However, I never make a diagnosis from the X-ray alone. I had the opportunity of studying over a hundred purulent maxillary sinus cases that I sent to X-ray. About 50 per cent of the X-ray diagnoses were correct. We must remember in looking at our X-ray plates that the bones of the two sides may be of a different thickness, or density, also that a thin watery pus will not give us the shadow that a heavy purulent pus will give and that polypoid degeneration will give very little, if any, shadow.

In my opinion, the safest and surest diagnosis of maxillary sinusitis is the Lichtwitz needle.

It is true there has been reported several deaths from needling the maxillary sinus and that on two occasions I have inflated the cheek muscles with air, but with proper instrumentation and due care I do not believe a patient runs as much danger of injury as they do while crossing a city street.

In the Ross H. Skillern Clinic, where not only the staff but also the students make daily needle punctures of the maxillary, no fatalities have ever occurred.

I believe if, after the needle is inserted, you withdraw the needle about half-way, then without undue pressure you slowly inject a small quantity of air into the sinus, you are subjecting your patient to little or no danger. If the sinus is infected you generally get a bubbling sound as the air passes through the exudate, but as I always wash out the sinus with warm normal salin solution once the needle is inserted, I depend not upon the sound, but on seeing the pus in the returned washings. Dr. Stout says that the washings from apparent purulent maxillary cases suffering with asthma often returns clear.

In a suspected case of maxillary sinusitis with a clear return of the washings, I generally have the patient return in a day or so, and again wash out the sinus. On several occasions, I have irrigated maxillary sinus with a clear return of the first, or even the second washings, to find a large gluttonous mass of thick, tenacious pus in the third washing. This pus has been so adherent to the mucus membrane that the fluid had passed over it, without dislodging it, or the mass was too large and firm to pass out of the ostium. After this pus was removed the patients stated their condition was greatly improved. With this kind of pus it is next to impossible to diagnose by posture or suction, until the contents of the sinus become thin enough to conform its shape to the ostium and run through this small opening, as brought out by Dr. Laessle.

In old chronic maxillary sinusitis in which the pus has a decided odor and so intimately mixes with the water as to form a milky fluid on its return, it shows a degeneration of the normal ciliated epithelium to either a polypoid mass or to pavement epithelium, and even though the bones may not be involved as yet, it will be, sooner or later, if radical procedures are not instigated.

There is such a case reported as cured after eighty some odd washings, but how many patients are going to allow that?

If after what I consider a sufficient number of washings there is still a foul smelling, milky-like return of the washings, I advise a radical operation, such as a perturbinal, Caldwell-Luc or Denker.

DR. LAESSLE: I consider this method to be valuable for certain types of cases, especially those of chronic sinusitis in children or adults, where the discharge is thin and where the ostium is large enough to permit the discharge; but in the acute stages and subacute, the rule is that you find the mucous membrane and the ostium so swollen that no discharge will take place. Now, in the chronic types, I can readily understand why this discharge will take place, but I do feel that the Lichtwitz needle is necessary, both from a diagnostic standpoint and a therapeutic standpoint.

C. P. LaG., age 30 years, real estate worker. Diagnosis: Paralaryngeal or extralaryngeal abscess; admitted Jefferson Hospital, Dec. 25, 1924; discharged Dec. 26, 1924; service Dr. P. S. Stout.

C.C.: Difficulty of speech, difficult swallowing. Pain in the neck.

F.H.: No bearing on the case.

P.H.: Typhoid fever when 15 years old. Influenza and pleurisy in 1922 (mild), from which the patient recovered and had no complications. Had tonsils removed eighteen months ago, after having had recurrent attacks of sore throat. Tonsils were said to be chronically infected. Had bronchitis ten years ago, also two years ago. Recovery uneventful. Had ankle fractured in 1912. Patient gives a history of

having convulsions during first three years of life. Has not had them since 3 years of age.

P.I.: Onset about one and one-half weeks ago with some difficulty in swallowing. Last Saturday (Dec. 20) patient felt chilly. Temperature that same night was 100° F. Temperature decreased on Monday morning and at noon of the same day the patient went to his downtown office. Also worked the next day (Tuesday). On Tuesday night his throat became sore and there was difficulty and pain when opening and closing the mouth. Iodin was applied on the side of the neck. Condition thought to be quinsy and the patient used "Lavoris". This morning (Thursday, Dec. 25) patient's condition became worse. Could not talk or swallow. Swelling apparently increased. Patient came to Dr. Stout, and upon examination and manipulation large amounts of a whitish pus was spat up by the patient. Throat condition immediately relieved. Could talk better and freely move jaws. Patient brought to hospital on afternoon of Dec. 25, Thursday.

Patient in bed, resting comfortably. No dyspnea, dysphagia or choking.

Head: Eyes and ears negative. *Mouth* m.m. of pharynx somewhat injected. Tonsils removed, tongue dark brown coating. Teeth in good condition. *Larynx:* Epiglottis and aryepiglottic folds edematous. Entrance to larynx very much decreased in size. Epiglottis so edematous that when first seen, looking with mirrors, not easily recognized. Right side of larynx swollen. Neck swollen.

Chest: Lungs: Breath sounds clear and regular. Expansion equal, no rales heard. *Heart:* Heart sounds are of good quality. No increase in heart dullness over normal. No murmurs, shocks, or thrills.

Abdomen: No pathology found.

Extremities: Negative.

Dec. 26, 1924, 1 p. m., upon discharge: *Larynx:* Epiglottis. Swelling and edema almost gone. Now able to see into the larynx. Cords not swollen or reddened, but appear short. Right side of larynx anteriorly appears slightly swollen or possibly pressed inward. There is no evidence now of abscess, but slight amount of pus just outside of larynx, right side. Swelling in neck gone and patient can eat, drink, and speak without pain or effort. Patient says he spat up pus during greater part of night.

Temperature rose to 101° on the evening of Dec. 25. Dropped to normal next day. Urine, negative. Sputum, negative for T.B. No other reports.

Additional notes: Appearance when I examined him at 12:30 p. m. on Thursday, Dec. 25, Christmas Day. Patient came into the office with his head held a little to one side, apparently suffering from something that was interfering with speech, swallowing, and beginning to interfere with breathing.

Examination showed some swelling on the outside of the neck, tender. Looking on the inside of the mouth no evidence of anything wrong as far as the appearance of the fauces was concerned, but with the laryngeal mirror very distinct and alarming symptoms were quite evident. The larynx was pushed over to the left, epiglottis swollen so that it was just possible to get a glimpse of the edematous arytenoids. A great mass was present on the outside of the larynx. While trying to see the extent of this mass it suddenly broke, pus was hawked up, about one ounce of white pus. Relief came at once.

The patient was sent to the Jefferson Hospital for observation.

DR. STOUT in closing: There was no salvation in this case. I am glad Dr. Kistler brought up his case.

We do not know where abscess opened. After it opened, he had a very high temperature.

DR. OLSHO: Posture the patient with head dependent and use the aspirating needle with suction, or incise, with suction in readiness.

DR. H. B. COHEN: About three years ago, I was called to the St. Agnes Hospital to perform an emergency tracheotomy. It was a patient of Dr. Laessle's and he could not get there as soon as I. All the history that I got was an abscess around the glottis, possibly gummatous. The

patient took a turn for the worse with marked dyspnea. Just as I entered the hospital I was informed that the patient felt something break and had brought up considerable pus. This gave him instant relief and no tracheotomy was done. A plus Wassermann was reported later.

I should like to ask Drs. Laessle and Stout whether they could or did find the site of the abscess.

DR. LAESSLE: I recall, many years ago, when I was a student, that Dr. J. C. Da Costa, in one of his lectures, emphasized the words: "Never operate upon a retropharyngeal abscess in an erect position." This made an impression upon me, and I suppose it appears like it does to all men when they start out to practice, that they seem to get the most severe cases at first, or at least, they appear to be the worst, and I can recall distinctly of having had the father suspend the child by the limbs, head down, and when the abscess was incised, huge quantities of pus was removed, and while draining, the head was lowered to avoid the pus entering either the lung or the stomach; fortunately, my case got well. Perhaps, Dr. Tucker can enlighten you a little more on this subject.

DR. KISTLER: About two years ago I was called to Philadelphia to visit a patient who had been under tear with laryngitis. Her breathing was very heavy.

Upon examination I found the right side of the larynx very edematous, epiglottis enlarged. Her breathing was so bad that I felt she would not live through the night, so I sent her to the hospital and had a tracheotomy tube inserted. I believe that if the patient had gone another twenty-four hours she would have died of suffocation. I watched her closely for about three days, and then brought into her room a tonsil suction machine and obtained about an ounce of pus around the abscess. Three days after the abscess was opened her recovery was eventful and her breathing was normal.

Paper. **Pemphigus as a Beginning Tonsillar Manifestation.** Dr. Henry Dintenfass and Dr. Sigmund S. Greenbaum.

Dr. Dintenfass in closing, in the diagnosis, which is of course of the utmost importance in this disease, there are three points to be considered.

1. The intense, out of all proportion to the clinical manifestations.
2. The great amount of salvation.
3. The fact that there is no glandular involvement. It is the absence of this involvement which differentiates pemphigus from syphilis and Vincent's angina, the diseases which it is almost similar to.

DR. S. B. GREENBAUM: Out of the twelve cases of pemphigus admitted to the Polyclinic Hospital in the last two years eleven died. One of the signs of malignancy of pemphigus is its onset with lesions on the mucous membrane. I have had a patient where the lesions were limited to the eye and mucous membrane of the palate. The blebs in the mouth rupture rapidly and there is left an erosion. I used in this patient mercurochrome intravenously. There is no definite cure known for pemphigus. The patient died, but not due to the fact that he was treated with mercurochrome, although that may have had something to do with it, but due to the fact that he refused to eat and felt that he was going to die.

More than 50 per cent of the cases begin with lesions in the mouth.

DR. WIEDER: I saw a patient about two years ago. For about two months he was treated for almost everything imaginable but pemphigus, the diagnosis being made only after the appearance of blebs on the body. He died within three months You have considerable redness of the mucous membrane in the areas in which the blebs have broken and the loose skin thrown off.

Meeting of April, 1925.

Paper: **Optic Neuropathy and Sphenoethmoid Disease.** Dr. Clifton M. Miller.

DISCUSSION.

DR. H. M. LANGDON: I wish to express my thanks to the Society for the compliment of permitting me to open the discussion on Dr. Miller's paper. It is a most complete exposition of the relation between the sinuses and the optic nerve to which he has limited his essay.

Inflammation of the optic nerve may occur any place in its course, but ophthalmoscopic changes do not occur in the early stages unless the portion involved is anterior to the entrance of the retinal vessels into the nerve, that is about 12 m.m. behind the globe. Inflammation behind this site is what we call retrobulbar neuritis and while we have loss of vision, especially central vision, and pain on moving the eye, especially if it be pressed back into the orbit with the hand, we do not have fundus changes until there is a descending atrophy with pallor of the temporal portion of the disc, from degeneration of the papillo-macula bundle. Should the neuritis occur anterior to the entrance of the retinal vessels we have edema of the disc and surrounding retina, with exudation of lymph and possibly hemorrhages from the pressure on the returning venous trunk. Both of these conditions may arise from sinus involvement but the retrobulbar type is by far the more common, resulting in a central scotoma, without changes in the peripheral field or the fundus.

Retrobulbar neuritis is also caused, as Dr. Miller has pointed out, by alcohol and tobacco, and is one of the most frequent complications of multiple sclerosis. Quinin ampbyopia results from a spasm of the retinal arteries, producing a degeneration of the ganglion cell layer of the retina and an ascending optic atrophy, with great diminution of the peripheral field of vision, but frequently normal central vision, a visual result quite unike that of retrobulbar neuritis. Where all of the more easily determined causes of retrobulbar neuritis are absent the sinuses are probably the cause, though other foci of infection may be the producing factor as was found in the case of:

Mrs. H., age 27 years, who reported in January last with a history that for six months O.D.V. had been blurred, without marked pain or inflammatory signs. She had been treated by one purporting to be an ophthalmologist, but no physical examination other than ocular had been made. Central vision was cut to 2/60 and there was a large absolute central scotoma with pallor of the temporal portion of the disc, on account of which a poor prognosis as to the return of central vision was given. She denied the use of tobacco and alcohol, there were none of the other manifestations of disseminated sclerosis and the nasal sinuses seemed the most likely place of trouble. Her rhinologist, Dr. George B. Wood, found no signs of nasal disease, but did find very badly infected tonsils, the removal of which resulted in a complete functional cure, central vision returning to better than normal, but the pallor of the temporal portion of the disc remaining.

The case was very instructive to me, being the first I had seen where retrobulbar neuritis resulted from infected tonsils and central vision returning to normal in spite of the apparent atrophy of the papillo-macular bundle.

Dr. Miller shows how more than one of the possible causes of retrobulbar neuritis may be present and I have no doubt that some of the cases where visual loss persisted in spite of the cessation of the use of tobacco and alcohol, were due to unrecognized sinus involvement, and possibly the other toxic agents were secondary or even not participants in causing the trouble, a thought pregnant with sadness when we consider the possibility of the needless self-denial.

To be Continued.